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PRACTICAL DIAGNOSIS:

THE USE OF SYMPTOMS IN THE DIAGNOSIS OF DISEASE.

BY

HOBART AMORY HARE, M.D., B.Sc.,

PROFESSOR OF THERAPEUTICS IN JEFFERSON MEDICAL COLLEGE OF PHILADELPHIA; PHYSICIAN TO THE JEFFERSON MEDICAL COLLEGE HOSPITAL; LAUREATE OF THE MEDICAL SOCIETY OF LONDON, OF THE ROYAL ACADEMY IN BELGIUM; CORRESPONDING FELLOW OF THE SOCIEDAD ESPAÑOL DE LA HIGIENE OF MADRID; MEMBER OF THE ASSOCIATION OF AMERICAN PHYSICIANS, ETC.

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PREFACE.

The object of this volume is to place before the physician and student the subject of medical diagnosis as it is met at the bed-side. To accomplish this the symptoms used in diagnosis are discussed first, and their application to determine the character of the disease follows. Thus, instead of describing locomotor ataxia or myelitis, there will be found in the chapter on the Feet and Legs a discussion of the various forms of and causes of paraplegia, so that a physician who is consulted by a paraplegic patient can in a few moments find the various causes of this condition and the differential diagnosis between each. So, in the chapter on the Tongue, its appearance in disease, both local and remote, is discussed. In other words, this book is written upon a plan quite the reverse of that commonly followed, for in the ordinary treatises on diagnosis the physician is forced to make a supposititious diagnosis, and, having done this, turn to his reference book and read the article dealing with the disease supposed to be present, when if the description fails to coincide with the symptoms of his case he must make another guess and read another article. In this book, however, the discovery of any marked symptom will lead directly to the diagnosis. Thus, if the patient is vomiting, in the chapter on Vomiting will be found its various causes and its diagnostic significance, and the differentiation of each form of this affection from another.

The value of the book is increased by the preparation of two indexes: one of symptoms and the other of diseases.

Basing his efforts upon the experience which he has had in both didactic and clinical teaching of large classes of students during the last twelve years, the author hopes that the work may in some degree lighten the labors of the general practitioner and student, and relieve the all-important subject of diagnosis of some of the difficulties which surround it. He has also endeavored to make the text serve as an aid to the rational use of his *Text-Book of Practical Therapeutics.*

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PRACTICAL DIAGNOSIS.

INTRODUCTION.

GENERAL DIAGNOSTIC CONSIDERATIONS.

A CLEAR understanding by the physician of the value of the symptoms of disease which he sees and of those described by the patient is of vital importance for the purposes of diagnosis and treatment, and one of the advantages of older physicians over their younger brethren is the ability which they have gained through long training to grasp the essential details of a case almost at their first glance at the patient. Much of this ability is unconsciously possessed because it is gained by a gradual process, yet it is none the less valuable, and its possession often impresses the patient with the insight which his physician has into his case. At first it is impossible for the novice to cast aside the minor symptoms, which the patient emphasizes as his major ones, and to perceive clearly that one or two facts that have been belittled in the narration of the story of the illness are in reality the stalk about which everything else in the case must be made to cluster.

Let us suppose the patient before the physician is one who has been able to walk into the office or dispensary. The attentive physician can at once gather much information about the case from the clothing, the gait, the build, the voice, the expression, and the manner. The thin man, with a peaked face and provided with an unusually warm overcoat, and still further wrapped up with a muffler almost to his eyes, is in all probability a sufferer from some pulmonary or throat difficulty, while the heavily built, phlegmatic individual, with a large head and well-filled paunch, is much more apt to suffer from gastro-intestinal or biliary catarrhs. Such a person will probably be one who habitually wears his coat open on the coldest days. Again, chronic drunkards, or persons whose mental powers are failing, often are exceedingly careless about their clothing, buttoning the coat or the trowsers with the wrong buttons, and

keeping the clothing dirty and spotted. Some cases of diabetes have first been discovered by the white spots on the trowsers, as the result of having allowed a few drops of urine to fall on the cloth, where they have dried. Old men who have incontinence of urine often wear trowsers which are stained in front, and they have often an ammoniacal odor about them from this cause.

The various forms of gait which indicate actual disease will be found discussed in the chapter on the legs and feet, but it may be mentioned in passing that in addition to these changes, which are dependent upon actual disease of the legs or the nervous system supplying them, that the general bearing and stride of a patient will often give us a clear idea of his general tone. The neurasthenic patient walks feebly or with a step that might be called ataxic, while the strong, hearty man of good physique strides along with a gait quite different from this, or that of an individual who is delicate and feeble.

Similarly, the patient's build betokens disease or health. The thin, tall, and hollow-chested person is recognized as a fair mark for the tubercle-bacillus, and the heavy, closely knit, phlegmatic man as one who may suffer from hepatic disorders. Again, the bearing of a person possessing a highly organized nervous system shows itself in the constant activity of his mind and body. No part is quiet for more than a moment, and drugs are more apt to produce extraordinary symptoms as the result of idiosyncrasies in this type of patient than in any other.

When the physician has gathered as much information as possible as to the age and general condition of his patient, by a careful scrutiny of his face and extremities, of which scrutiny, however, the patient should be unconscious, he should ask him to tell what brings him for advice, and as a rule this will be the opportunity the sufferer seeks to pour out the story of his ailment as he sees or feels it. Often the story will seem wearisome, and, to the educated mind of the physician, wandering or unnecessary; but to the patient every word seems of the greatest importance, and to show any lack of interest may give the impression of carelessness, or it may interrupt the story just as a most important symptom is about to be described. Even if the patient is unable to convey a very clear idea of his condition, the manner in which his story is told, the character of his speech, and the expression of his face while speaking may give useful information as to his ailment or general state.

If, instead of the patient being an office or dispensary case, he is one who is being visited at home, the fact that the patient meets the physician in one of the living rooms rather than in a bedroom indicates either that there is little immediate danger in the case, or, at least, that the difficulty is not acute, but chronic in type, as some slowly progressing form of pulmonary, cardiac, or renal diease. Of course, there are exceptions to this rule, as in the case of a patient who, having caught a heavy cold, is remaining indoors, but not in bed, for prudence' sake. Or, again, if on seeing the patient we find him sitting in a chair only partly dressed and propped up with pillows, or instead leaning forward upon the back of a chair placed in front of him, we know that he is the subject most probably of an acute or chronic heart disease, most likely an acute exacerbation of the latter. A glance at the face of such a patient, revealing a trembling nostril, blue lips, or an anxious facies, will aid still further in directing attention to the heart or lungs, and the hands if examined will appear relaxed and livid or darkened in hue, indicating capillary stasis and deficient oxidation. In other cases, however, the patient found sitting propped up with pillows may be a convalescent from some long illness; but if so, the general atmosphere of the patient is better, and the surroundings are apt to be more tidy.

If we find the patient in bed, he may be lying abnormally quiet as the result of faintness or acute nausea, or, perhaps, from partial or complete coma due to cerebral or renal disease, or from the effects of some drug; or, again, he may be rolling about the bed from the pain of acute belly-ache, or be keeping his legs and body very still while his hands and head are ever on the move to prevent anything from suddenly approaching or touching his abdominal wall, as in peritonitis. The striking difference between the activity of the head and the fixation of the lower part of the body, in peritonitis, is notable. Sometimes, however, anxious restlessness indicates acute internal or external hemorrhage, but here the movements are minute though active, and the patient does not expend much strength as he does when suffering from pain. Usually a patient who is lying on his side turns on his back as the physician or nurse approaches, in order to face his visitor; but if he persistently remains on the side without moving except to turn his head partly, we may suspect that in that posture he is most comfortable, and that the position is assumed for its comfort or to relieve pain or dyspnœa.

Thus, in acute pleurisy the patient lies with the affected side uppermost because it is too sore to permit him to touch it to the bed; whereas, if the stage of effusion has arrived, he lies on the affected side in order to give the side which is healthy free play in compensatory respiratory movements, and to remove the pressure of the effusion from the healthy lung. If the patient lying in this posture is not suffering from pleurisy, his position may be assumed to be due to an effort to relieve the discomfort caused by an enlarged liver. The fact that the patient lies constantly on the back is also a characteristic of grave and advanced disease in some instances. Very ill persons almost never lie on the side, and the fact that a desperately ill case of yesterday is found lying on the side to-day is an encouraging sign. Persons with severe heart disease are rarely, if ever, able to lie prone in bed, and have to be more or less propped up with bed-rests and pillows. Large growths in the abdominal cavity producing pressure on the diaphragm also necessitate this semi-prone posture, and double pleural effusions, or pulmonary consolidation, or œdema, produce the half-reclining attitude in order that the upper parts of the lung may be used to advantage.

Again, if the patient wakes when spoken to, and then drops off to sleep at once, some form of poisoning may be present, as from opium, or the poison of advanced hepatic or renal cirrhosis may be present. (For the significance of picking at the bed-clothes, see chapter on Hand and Arm.)

We can next pass to a consideration of the objects to be sought in questioning a patient as to the illness from which he is suffering. Often much information can be gained by a well-directed question, and a favorable impression can be made upon the patient by the manner in which it is put and the bearing which it has on his case. Thus, if a man is evidently much emaciated, and his clothes fit him loosely, a question in regard to his loss of flesh is very appropriate; but if he is manifestly too stout for comfort such a question will be most unfortunate. Or, again, if a young married woman comes complaining of constant sickness of the stomach and a fanciful appetite, and the physician directs all his questions to the condition of the stomach without an eye to a slight increase in size about the waist or below it, his professional acumen is in grave danger of being libelled by that same woman, who knows, or soon finds out, that her discomfort is due to pregnancy.

If the woman is unmarried and there is no evidence of gastric

disorder on her tongue, it is well to remember what Battey, of Georgia, said in regard to this condition: "Always believe a young unmarried woman with abdominal tumor, of high social position and unimpeachable virtue, if she has been watched over by a platonic and abstemious young cousin of the male persuasion while the mother went out, to be pregnant."

Again, if a married woman of some years tells her physician that she has no children, the physician naturally asks some questions which elicit the fact that she has had frequent miscarriages. He in this way finds quite as much about probable syphilitic infection as if the question had been put: "Have you ever had a sore on your privates," which would embarrass the patient, produce domestic troubles, and probably be lied about if she was forced to answer the question.

Again, when asking a woman about the health of the living parents, or the cause of death of the dead, care should be taken not to ask a direct question, as, for example, whether the mother has died of cancer, for the patient may be already greatly worried lest she has that disease. It is better to ask the cause of death, or of the illness she is suffering from. If the story is that the parents died of "bronchitis," in all human probability the real cause of death was tuberculosis of the lungs.

If the patient complains of pain, past or present, the best way in which to discover its true seat is to ask him to place his hand on the affected part, as in this way errors in his description of his anatomy will not be committed, and false impressions will not be conveyed to the physician's mind. Even this direct method of showing the area of pain is not to be absolutely relied upon, for often pains are referred to parts in which there is no disease. Thus, the pain of coxalgia is apt to be felt in the knee and ankle, and in children the pain of acute pulmonary disease is often described by the patient as felt in the abdomen. If the pain has been really abdominal, there will, in most cases, have been diarrhœa or free passage of flatus. It is not to be forgotten, on the other hand, that a question which discovers the fact of several movements of the bowels does not prove the presence of true diarrhœa, because a purgative may have been taken by the patient.

In asking questions as to constipation the physician must not forget that the opinion of the patient as to what constitutes regularity of bowel-movement is of very little value in many instances. A

daily movement is not known to many patients, and a movement every few days may be quite sufficient to justify the statement, in their opinion, that no constipation is present.

The young physician, in particular, in asking questions of women patients of the better class, should not hesitate to ask direct questions as to the state of the bowels or of the menstrual function. To hesitate or ask indirect questions about such matters simply produces embarrassment, which otherwise would not exist, and intimates that the question is one of doubtful propriety, when in reality it is most important and proper.

If the patient to be examined is a child, it is well for the physician to remember that his mere presence as a stranger may be a source of alarm, and that the association in the child's mind of sickness and the doctor, and badly tasting medicines, is sufficient to render him a much-to-be-dreaded individual. Generally it is best, on entering the room where the child is, to pretend to pay no attention to it whatever, but to engage in conversation with the mother or other person, speaking of the case in a way which the child will not understand. Very often this very lack of attention will result in the child forcing the recognition of his presence upon the physician by making the first advances toward friendship, and this is particularly apt to be the case if the child is already spoiled by over-attention by the family and friends. Time should always be given the child to grow accustomed to the peculiarities of the visitor, and if any instrument for diagnosis is to be employed, it is best to hold it in the hand as if it were a plaything before attempting to put it into actual use. The tact which the physician must exercise in diverting a sick child is an essential to the successful treatment of children. Some physicians are welcomed to a house by the sick and well as a Santa Claus would be, and others, devoid of the trait of amusing children, are fled from as if they were dragons.

During the time that the physician is allowing the child to get accustomed to his presence he should be gaining much useful information about the case by observing the movements and expression of the child; its color, size, nutrition, breathing; the shape and size of its head; the condition of the lips, whether moist or dry; and, if the child is speaking, the tone of its voice, or, if crying, the character of its cry. It is needless to state that a child may cry from fright, from pain, anger, or hunger. Constant screaming crying is, however, nearly always due to the pain of earache or hunger, for

abdominal colic is usually intermittent. If there be pain in the ear, the hand will often be rubbed over the affected side of the head, and the child will not be pacified by the offer of the breast. If the child coughs, and then begins to cry, pneumonia or pleurisy may be present; or in other cases the pain is so great that the child is cryless. A sharp, piercing shriek of crying indicates the pain of meningitis in many cases.

If the crying child be placed at the breast, which it takes with avidity only to drop the nipple in a moment with a cry of pain or anger, one of two conditions is present: either the child has stomatitis or the breast is empty; or, again, if it seizes the breast and then lets go with a gasp, it probably has coryza, or syphilitic snuffles, which prevents it from breathing through the nose while sucking. Similar signs may be present in any other condition producing shortness of breath.

If a child over four months of age cries and sheds no tears in the course of an illness, this is an unfavorable sign.

It is important to notice whether there is languor or a tendency to play. A healthy infant, when awake and well-fed, is always kicking or cooing and moving its arms about, and has a happy expression on its face; whereas if any cerebral trouble is present, it often has an anxious frown, or its hands are placed to the side of its head or rubbed over the vertex.

In a perfectly healthy child which is sleeping the respiration should be practically inaudible, and it is a good practice to note the regularity of the breathing in all patients while they are asleep, as it is then unaffected by voluntary effort. In children a sighing breathing, or one disturbed in rhythm, indicates a disturbed digestion or fever.

The breath of the healthy child is invariably odorless and sweet, but is apt to become heavy and sour in fever and gastric disorders, and in tonsillitis and diphtheria it is apt to have a peculiar sickening odor of a sweet character. In cases of empyema opening into a bronchus or in gangrenous stomatitis the breath is very offensive.

The physician should also, by careful questioning of the nurse or mother, find out how long the illness has lasted, the manner in which it began, the fact as to whether a similar attack has occurred before in this or other children of the family, and the state of the temper, appetite, bowels, and urine of the patient, for an irritable temper in a child means ill-health, as does also a poor appetite, constipation, diarrhœa, or abnormal urine.

The expression of the face, shape of the head, and similar noteworthy points in the diagnosis of the case will be more thoroughly discussed in the chapter devoted to these parts.

When it comes to a close examination of the child, great care must be exercised. The character and rapidity of the respirations are best studied at a distance before excitement has disturbed them, and the best way of listening to a young child's chest is when it is held over the shoulder of the mother as if she were carrying it for a walk, or, if the child can be taken in the physician's arms, its buttocks should rest on one hand, while its chest leans against the other. In this way the physician can listen to the back of the chest without difficulty, keeping the child amused by walking up and down the room while it is in his arms.

If it is not possible by any bribe to cause the child to protrude the tongue for examination, the physician will often be able to see this organ when the mouth is widely opened in crying. In taking a child's pulse it is best to take it while it is asleep, if possible, as the excitement of the physician's visit or the crying on awakening will greatly increase the pulse-rate.

PART I.

THE MANIFESTATION OF DISEASE IN ORGANS.

CHAPTER I.

THE FACE AND HEAD.

The expression and color of the face—Facial paralysis, unilateral and bilateral—Ptosis—Facial spasm—The shape of the head—The movements and position of the head.

So much can be learned by the physician from the expression and general appearance of a patient's face and the carriage and shape of his head, that a careful inspection of these parts should always be made. For this reason, in the consulting-room and at the bedside, the physician should always arrange his chair in such a way that the light falls upon the face of his patient, while his own is in the shadow, and this is of importance not only because the facial expression of the patient can thus be well seen, but also because it prevents the patient from making a too close scrutiny of the physician's face with the object of detecting encouragement, lack of sympathy, or alarm.

The Face.

The Expression is produced by the formation of creases, or alterations in the contour of the skin and subcutaneous tissues by trophic and muscular action, and these changes are in time brought about by the mental tendencies and habits of the patient, his temperament, his intellectual development, his exposure to outdoor or indoor influences, and finally, and these are very important, by pathological processes which may be going on somewhere in his body. The temper of the man also affects his expression, particularly as he approaches middle life, and he looks amiable, capable of sudden anger, or sullen, as the case may be.

The intellectual face is easily recognized. Sometimes it is deeply thoughtful and placid, at others eager or keenly alive to the surroundings or the conversation, and it separates the man descended from several generations of men who have lived as thinkers from him whose ancestors have been but recently wage-earners by physical labor, involving only ordinary human intelligence.

Fulness of the lips, particularly of the lower lip, is supposed to be present in persons of strong sexual appetite, and often indicates a phlegmatic temperament, whereas the thin, mobile lip is typical of the high-strung, nervous individual.

The expression of the lips as a whole is also to be regarded in connection with the expression of smiling. The risus sardonicus of strychnine-poisoning or tetanus is quite characteristic, and the simple smile of hysteria is equally notorious.

The skin of the face and the expression about the eyes of one who has been exposed for years to the weather are so characteristic as to need no description, while the face of the clerk, whose life is almost entirely spent indoors, is pale and wan.

Similarly, the face of a person who uses alcohol to excess is generally flushed, heavy, and more or less expressionless. The eyelids are reddened more than normal, and the skin is apt to be puffy and unhealthy-looking. Women at the menstrual period, or when suffering from menstrual disorders, often have dark areas under the eyes, and pigmentation of the eyelids is often seen in females very early in pregnancy.

The color of the face is discussed in the chapter on the Skin, but it is not out of place to note at this point the pallor of the face in fright, faintness from hemorrhage, acute or chronic, that due to lack of proper food, and the peculiar pallor of chlorosis. In the latter disease the faint yellowish-green tinge of the skin in some parts of the face, which still retains its plumpness, is quite typical. A parchment-like skin stretched over the face so that it appears as if dried over the under-structures is seen in some young persons suffering from syphilis, and in some cases of alcoholic hepatic cirrhosis.

The color of the face may be rendered gray or bluish by the ingestion of overdoses of the coal-tar products, such as acetanilide, antipyrin, and phenacetin, and it is curious that this effect is best seen when the patient is viewed at a little distance.

(For the indications of facial cyanosis, see chapter on the Skin.)

In view of the extraordinary variations seen in the expression of

the face in the healthy it is not surprising that this part of the body should give the physician, when studying disease, so much useful information. It is an interesting fact, too, and one not unworthy of note, that the true facial expression of a disease is rarely aped by a malingerer, and in all diseases is unrecognized by the patient even though he sees himself several times daily in the looking-glass. Thus it is by no means uncommon to see a person, who is suffering from the onset of some sudden and grave disease, bearing upon his face what we call "an expression of anxiety," when he himself as yet has no conception of the gravity of his illness. This expression is very characteristic of serious illness, and, though difficult to describe, when recognized becomes quite valuable as a diagnostic factor, particularly as it rarely, if ever, is exaggerated by the patient who bears it. It is seen most markedly in cases of severe acute croupous pneumonia or peritonitis, or after severe injuries.

When persons have had continuous pain for a long time, as in patients who have growths of a malignant character or other organic disease, the expression of the face, naturally gentle, often becomes hard and stony, or, if the pain be in the head, the expression is not only that of pain, but of profound mental depression. In cases of carcinoma the face becomes thin, its skin yellow and straw-colored, and oftentimes greasy and thick, and there is often a marked look of anxiety. On the other hand, the patient sometimes has a dogged expression on his face as if he had been told of the true cause of his illness, and was rebelling against the inevitable progress of the disease.

In the case of children, much information can be gained as to the state of the body by the facial expression, particularly while the child sleeps. If it is asleep and healthy and well, the eyelids are closed, the lips are ever so slightly parted, the nostrils are practically immobile, and the general expression is very peaceful. If, on the other hand, the eyelids of a sleeping child are slightly parted so as to show the whites of the eyes, there is present some digestive or nervous disturbance, perhaps accompanied by moderate pain. If the disease is grave and the eyelids remain far enough apart to result in glazing of the conjunctiva from dryness, this is a sign of grave import. Again, twitching of the eyelids often indicates nervous irritation or the early stages of the convulsive state, and it is not uncommon for an expression to pass over the face of a child who, while sleeping, is suffering from pain, which begins as a smile and ends with a drawing-in of the corners of the mouth, an expression somewhat

like that seen on the face of a waking child when it seems to be in doubt as to whether to laugh or cry. Whether asleep or awake a child in pain, if not crying, has a pinched look about its nose and mouth, and sometimes some idea of the seat of the pain may be gained by the part of the face which is drawn. When pain is in the head, the forehead is apt to be wrinkled into a frown; if the nose is pinched and drawn, it is said to show that the pain is in the chest; and if the upper lip is raised, pain is probably felt in the belly. Aside from these symptomatic manifestations, however, we find in the face of a child several evidences of important diathetic tendencies, or even hereditary diseases. Thus we see the light flaxen-haired, slimly built child with a refined *spirituelle* face and transparent skin, whose temporal veins can be easily traced and whose expression is often thoughtful and deep. Such a child often comes of tubercular parents, and is frequently a victim of tuberculosis, in one of its rapid forms, as it approaches puberty. Or, again, the child is heavy and cheesy-looking, apparently solid and sturdy, but its features are heavy or perhaps even coarse, while its neck is thick and short. Such a child is often a victim of tubercular bone disease. In other instances, a square projecting forehead with faulty bone-development elsewhere indicates rickets, or an immense bulging forehead with a wizened, puny face beneath shows hydrocephalic tendencies. Sometimes a broadness of the bridge of the nose or marked flatness of it indicates congenital syphilis. Such a child is often much wasted, its features pinched, and its lips thin, while the flattened nasal bridge is bluish and its face is often that of a little old man, shrivelled and wrinkled. Mucous patches at the corners of the mouth or around the anus are often found in such cases, and soon confirm the diagnosis of infantile syphilis. Finally, in respect to facial expression in childhood, attention must be called to the "fish mouth," vacuous, and "nose-pinched" expression of those children who are "mouth-breathers" from nasal obstruction. (Fig. 1.) Great immobility of the lips and cheeks may be due to mucous patches or other ulcerations of the buccal mucous membrane, and if high fever is present the presence of herpetic blisters about the lips points to croupous pneumonia in the child or adult.

In adults the facial expression of many diseases is even more characteristic than it is in children. Thus we see in acute pulmonary phthisis the widely opened eye, the hunted expression, the quivering nostrils, the red flush over the malar bones, the wasting

and dryness of the hair and skin, and the eager, or apathetic, glance of the eye.

In severe pneumonia the flushed face, with a deeper red on one cheek than the other, the anxious expression, and the dilated nostril are noteworthy; and in the dyspnœa of heart disease the dilated nostril and constant opening of the mouth as if seeking for air, with the facial pallor or cyanosis, are characteristic. Often, too, in chronic cardiac or pulmonary disease producing slight difficulty in respiration, the patient's lips are seen to be slightly parted and dry, and often appear somewhat cyanotic. In children suffering from lesions of the mitral valve of the heart it is very common for some blurring or indistinctness of the features to be present.

FIG 1.

Boy aged seven. Mouth-breather, from obstruction of the pharynx; open mouth; vacant expression; pinched nostrils; dull eyes; drooping eyelids; sunken chest; round shoulders. (HOOPER.)

One of the most characteristic facial expressions that we meet with is that of typhoid fever or fevers of a typhoid type. The face is dull and expressionless; the teeth are covered by sordes, which become brown and blackish by exposure or by discoloration from medicines and foods; the lips are often moved in a low muttering delirium; and the whole appearance is that of apathy. Even when spoken to, the face of a patient suffering from enteric fever rarely lights up in response to the greeting.

Equally, if not more, characteristic is the facial expression of acute peritonitis. The upper lip is drawn up in such a way as to show the teeth, and the expression of anxiety and nervous unrest is well developed. Similarly in abdominal pain due to other causes than peritonitis there is often a twitching of the muscles of the lip and about the eye which is quite typical. This twitch is said by

Fothergill to be peculiar to pain below the diaphragm, and he is also responsible for the statement that it is best seen in the face of the parturient woman in the second stage of labor.

The facial expression of hysteria may be apathetic, or it is that of devotion, rage, or grief, and these expressions are fixed if the patient be cataleptic. If she is not cataleptic, not infrequently one expression may succeed the other, or in their place there comes that curious smile or vacuous expression of the face which is so characteristic. It should be remembered, however, that this vacant, fatuous look may occur in women suffering from the early stages of disseminated sclerosis and in children with chorea. Then we have the elated facial expression of general paralysis of the insane, the excited look of acute mania, the beaten, weary, careworn look or apathetic glance of nervous exhaustion, and the hopeless expression of melancholia.

The face of paralysis agitans is sometimes called the "Parkinsonian visage," is distressed and pathetic, and yet somewhat intense. (See chapter on Hand and Arm, part of on Tremor.)

A pale, puffy face, generally looking worn and weary, may be seen in cases of chronic or subacute renal disease. In children there is often in this condition a peculiar transparent or pearly look in the lower eyelid, so that it seems somewhat pellucid. Great swelling or œdema of the face is seen in erysipelas, dropsy, and inflammatory swelling (see chapter on the Skin). In trichiniasis the eyelids are often swollen early in the disease, and then recover their normal appearance only to become swollen again later in the malady.

When the face bears a sleepy, listless expression, the forehead being devoid of wrinkles, and there are present faulty movements of the lips, which cannot be approximated, as in whistling, and at the same time the patient is unable to close the eyes entirely, although the lids droop, the physician should think of the possibility of these being the early symptoms of what has been called the "facio-humero-scapular" type of muscular atrophy (Landouzy and Déjérine). This disease, as its name implies, speedily involves the scapulæ and arms after affecting the face, and exophthalmos is often present. This form of muscular atrophy lacks the fibrillary twitchings seen in spinal progressive muscular atrophy; there are no changes in electrical excitability, except that owing to the loss of muscle-fibre the reaction is feeble. The fact that more than one member of the family is affected, and the long duration of the disease added to these signs, render the diagnosis easy. It is a rare disease.

An appearance of the face almost identical with that just described is seen in Friedreich's ataxia, and is often one of the earlier manifestations of the disease; but the presence in Friedreich's ataxia of the ataxic gait, the jerky articulation, nystagmus, loss of knee-jerks, and absence of muscular atrophy separate it from the Landouzy-Déjérine type of muscular atrophy just described as facio-humero-scapular atrophy.

FIG. 2.

Cretin. (DERCUM.)

The facial expression of cretinism is exceedingly characteristic. The nose is broad and flat, the eyelids swollen, the lips are greatly thickened, and the enlarged tongue lolls out of the mouth, from which saliva constantly dribbles, while the waxy skin and subnormal temperature of the body, with a poor circulation, slow respiration, and mental hebetude, complete the symptom-group. There is nearly always in well-developed cases marked lumbar lordosis. (Fig. 2.)

In certain forms of leprosy the face often becomes leontine or lion-like in appearance.

The facies of exhausting disease about to produce death is very characteristic, and is seen frequently in cholera and in tuberculosis of the lungs. It is called the "Hippocratic face," and is peculiar in the sinking-in of the temples where the jaw-muscles are inserted; the eye is sunken, and around it are great hollows, so that the infra- and supra-orbital ridges become greatly accentuated. The eyelids are slightly parted, the cornea somewhat glazed; the nose pinched, its skin drawn; and the lower jaw somewhat dropped. Such a face, if typical, is a sure forerunner of dissolution.

Facial Asymmetry. Facial asymmetry is sometimes seen as a congenital defect, and curiously enough is often developed in children who suffer from congenital wry-neck. This is not to be confused with that extraordinary affection called facial hemiatrophy, which usually begins in childhood in one spot, and slowly proceeds until one side of the face, sharply outlined from the other, becomes wasted in its skin, muscles, bones, color, and hair. Even the eye may be sunken and shrunken. Rarely this wasting is bilateral.

Sometimes in facial hemiatrophy the wasting is accompanied by painful twitchings, which increase with mental excitement. More rarely there is decrease in the acuity of taste and hearing on the affected side, while myosis, sweating, or excessive dryness of the skin may be found on this side. Such symptoms as the last show involvement of the sympathetic nerve-fibres. The changes are probably due to disease of the fifth (trifacial) nerve.

As to whether circumscribed scleroderma (morphœa) and facial hemiatrophy are identical, that is, whether the first is a well-developed form of the latter, is not decided. Hyde apparently regards them as identical. (See chapter on Skin, Scleroderma.)

Even more rare than facial hemiatrophy is facial hemihypertrophy, one side remaining normal in size and the other becoming gigantic.

The massive face of a person suffering from acromegaly is very characteristic (Fig. 3), and the face has a full-moon broadness in myxœdema. The face in osteitis deformans is shaped like a triangle with the base upward. The enlargement of the bony parts of the skeleton, the kyphosis, and the comparative muscular feebleness of acromegaly aid in the diagnosis of that disease, for in myxœdema there is no true bony enlargement. In osteitis deformans the shafts of the long bones become weakened, and their surfaces roughened from periosteal deposits.

nerve-trunk or foramen, is when there is a tumor at the base of the brain involving the facial fibres below the facial nucleus or destroying the nucleus itself.

Very rarely in cerebral facial paralysis is the loss of power as complete as it is in the peripheral form. Again, in cerebral facial paralysis the eye on the paralyzed side can usually be closed and the forehead wrinkled, whereas in the peripheral form it cannot. Why this should be so is not clear, unless it is that in the muscles used commonly in pairs, as in those of the forehead, there is an adequate nerve-supply through direct non-decussating tracts which innervate the muscles. When facial paralysis has associated with it none of the signs of peripheral wasting, and none of the remote causes of hemorrhage, embolism or thrombosis, such as result from impaired blood-vessels or a diseased heart, and when the paralysis comes on gradually (though it may be sudden from surrounding inflammation), the condition is probably due to cerebral tumor. This diagnosis is confirmed by the gradual spread of the paralysis to other parts, as the arm and then the leg on the same side of the body, and by the development, often before each spread of the paralysis, of a convulsion. The facial paralysis resulting from tumor at the base of the brain differs from that due to cerebral tumor or hemorrhage by the fact, already stated, that the reaction of degeneration quickly develops in the paralyzed part; that the parts supplied by the frontal branch of the facial are often quite as much paralyzed as are those supplied by the lower branch, which is rare in the cerebral lesion, and there will commonly be found other evidences of a growth which, in a region so densely filled with important centres, speedily affects other functions. Thus, there will nearly always be found in association with this form of facial paralysis paralysis of the oculo-motor and abducens, causing ptosis, a moderately dilated pupil, and internal or external squint. The optic nerve may show choked disk, and there may be disturbance of vision (see chapter on Eye). If the tumor grows large enough, or is so placed as to involve the facial fibres for both sides as well as those of the oculo-motor, abducens, and optic nerves on both sides, all these symptoms become, of course, bilateral.

Facial palsy associated with deafness may indicate cerebellar tumor, the diagnosis of this cause being decided by the other cerebellar symptoms, such as the peculiar gait. (See chapter on Feet and Legs.) Such growths are not uncommon in children.

Sometimes very shortly after birth the child is seen to have a

facial paralysis resulting from pressure by the forceps, which have slipped and injured the nerve, or have caused an extravasation of blood into the neighborhood of the parotid gland, thereby causing pressure on the nerve. The prognosis is usually favorable if due to such causes; but if the forceps have caused facial palsy by producing a cerebral hemorrhage, the outlook is bad.

The possibility of facial paralysis being due to hysteria should not be forgotten. The loss of power under these conditions may be unilateral or bilateral, generally the former. Its association with the symptoms of hysteria described in the chapter on the skin, and elsewhere, in this book will aid in making the diagnosis.

There yet remain to be considered several forms of facial paralysis unilateral in character yet associated with paralysis elsewhere.

Unilateral facial paralysis very rarely occurs in association with monoplegia in acute anterior poliomyelitis. So seldom does it occur in this connection that it has been denied an existence. Often it is but temporary, while the monoplegia of the arm is permanent. It occurs more commonly in the disease in adults than in children.

Facial paralysis with arm paralysis of the same side, followed in a short time by paralysis of the leg of the opposite side, is quite a characteristic symptom of syphilitic arteritis at the base of the brain.

Crossed paralysis—that is, paralysis of the face on one side, and of the arm and leg on the other—is due to a lesion in the pons above the decussation of the pyramids and below that of the facial fibres. (Fig. 4.) Thus it is seen in this figure, on the left side, third inscription, that the lesion in the pons cuts off the motor fibres in the place indicated, thereby causing the distribution of the paralysis just named. (See also chapters on Hemiplegia and on Arm and Hand.)

Sometimes the muscles supplied by the facial nerve escape paralysis, but those of the jaw, namely, the masseters and temporals, become paralyzed either bilaterally or more commonly unilaterally. This is a rare affection, and depends upon paralysis of the inferior maxillary branch of the trifacial nerve. This may be due to pressure produced by growths or inflammatory processes at the base of the skull. It may also occur as the result of hemorrhage into the medulla, or from progressive bulbar paralysis.

Ptosis. In connection with the subject of facial paralysis that of ptosis or drooping of the upper eyelid must be considered. It depends upon loss of function of the oculo-motor nerve or its centre or nuclei. (Fig. 5.) It is a symptom of the greatest importance,

first, because it is so readily recognized; second, because it is a source of great annoyance and alarm to the patient; and, third, and more important, it often gives us very clear ideas of the condition of the patient. The presence of this symptom should call to the physician's mind the various causes which produce it.

FIG. 4.

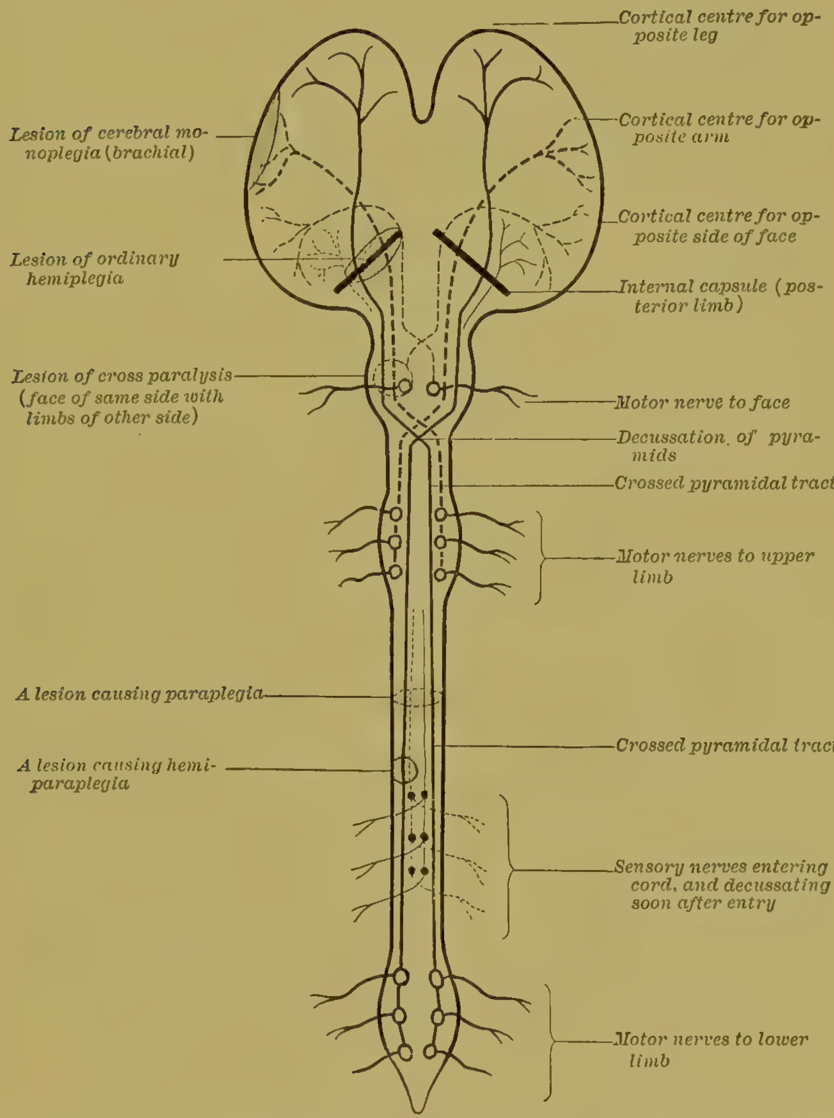


Diagram to show the general arrangement of the motor tract and the effect of lesions at various points. (ORMEROD.)

In the first place, it sometimes occurs as a congential defect, and in such a case the history of the patient renders the diagnosis easy. Second, it depends upon a lesion of the oculo-motor nerve or its nucleus. If this nerve be entirely destroyed so far as its function is

concerned, there will be, in addition to ptosis, paralysis of all the external muscles of the eye except the superior oblique and external rectus, and in addition there will be a moderately dilated pupil, which will not contract, and paralysis of the ciliary muscle—that is, loss of accommodation. The eye can be moved outward by the action of the external rectus, and a little downward and inward by the superior oblique. Diplopia is present, and a little exophthalmos

FIG. 5.

Ptosis in a case of alternate hemiplegia of syphilitic origin. (Philadelphia Hospital.) (DERCUM.)

may be present owing to the action of the superior oblique, which presses on the ball. If the lesion be in the oculo-motor nucleus, the near position of the nuclei of the fourth and sixth nerves will probably cause them to be affected also, thereby causing a general ophthalmoplegia. If the lesion is not nuclear, it may be due to disease in the nerve itself, as already pointed out. If this is the case, the lesion is probably due to pressure in the cavernous sinus or periostitis of the bones forming the sphenoidal fissure through which the nerve passes. Sometimes, however, the paralysis of the nerve may be only partial, so that the external muscles of the eyeball escape, and only ptosis and a dilated pupil are present. Very rarely ptosis results from a cerebral hemorrhage, without the other signs of oculo-motor paralysis being present. That is to say, the branch of the oculo-motor which supplies the levator palpebraris is affected, while the branches supplying the external and internal ocular muscles escape.

If there is a history of a cerebral attack resembling a mild apoplexy, and a unilateral ptosis is present, the lesion is probably in the cortical centre for the oculo-motor nerve in the angular gyrus just below the

inter-parietal fissure. The lesion is, of course, upon the opposite side of the cortex from the ptosis. Such a case is very rare.

A fourth cause of ptosis is due to an affection of the sympathetic nerve, and is sometimes called pseudo-ptosis. There are associated symptoms of vascular dilatation, with redness and swelling of the skin of the affected side, elevation of temperature in that part, contraction of the pupil on the affected side, and apparent shrinkage of the eye into the orbit. This form of ptosis results from the paralysis of the unstriped muscular fibres of Müller which exist in the orbital fascia, for as these muscular fibres aid in holding open the lid their paralysis results in partial ptosis. Nothnagel asserts that such symptoms occur with lesions in the corpus striatum.

A fifth cause of ptosis is reflex irritation usually through the fifth nerve. This is probably due to an inhibition of the oculo-motor centre. It is usually only transient.

Sixthly, it is not uncommon in cases of nervous syphilis for so-called alternate ptosis to develop. First, one eye is affected by ptosis, and then the other just as the first begins to improve or has recovered.

Ptosis has been known to complicate tetanus, probably as the result of reflex irritation of the fifth nerve.

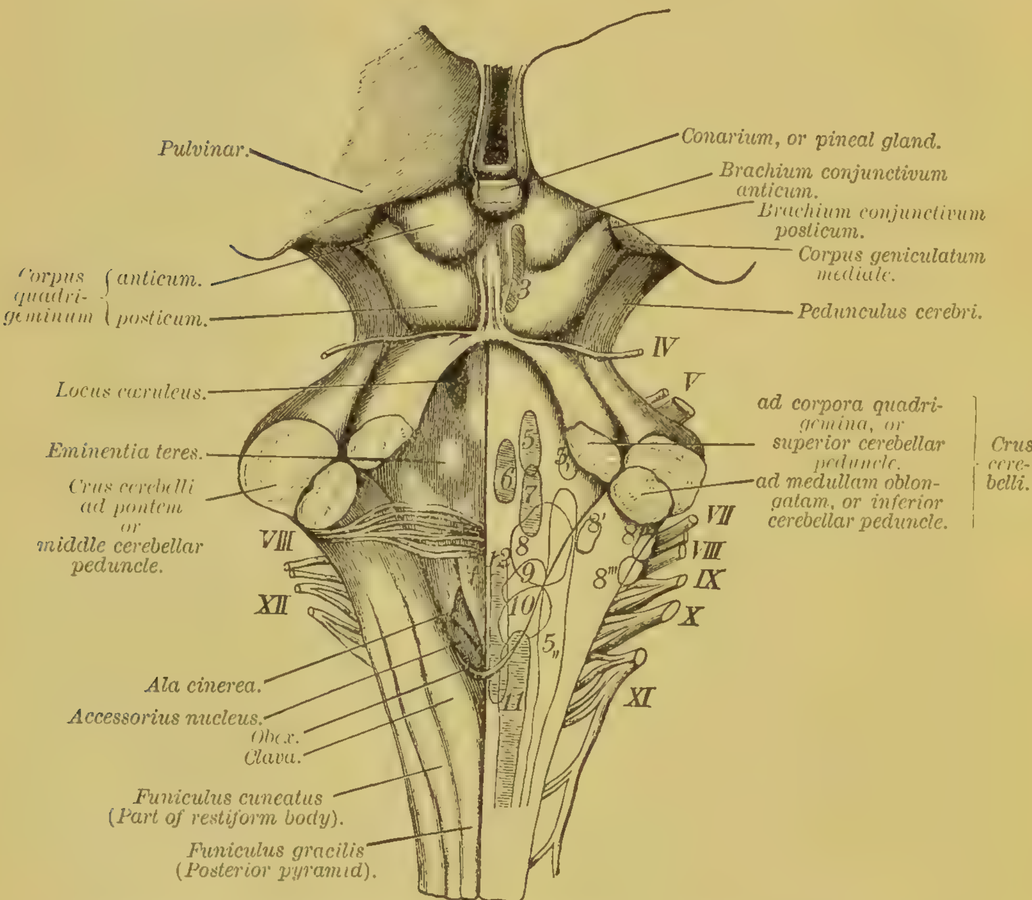
Ptosis, either unilateral or bilateral, may arise from hysteria and idiopathic muscular atrophy. If from hysteria, the diagnosis can be made from the age, sex, and history of the patient, from the presence of the hysterical sensory changes described in the chapter on the skin, and from the fact that there is a tendency to spasm of the orbicularis muscle when the patient is made to look up. This contraction of the orbiculares proves that there is no true paralysis of the levators. If the ptosis is bilateral and hysterical, the head is tipped back when the patient is told to look up.

Single or double ptosis is by no means a rare symptom of locomotor ataxia, and is often associated with other evidences of oculomotor palsy. Sometimes diplopia due to these changes are the first symptoms complained of, and the patient may state that the diplopia comes and goes.

Bilateral ptosis may arise from tubercular or syphilitic changes about the base of the brain, or it may be congenital, or if transient be caused by poisoning by gelsemium or conium. It is also seen in feeble, overworked women, particularly in the early morning on awakening.

Again, it is not very rare to see slight drooping of both lids in all the members of a family, in which case the condition is usually most marked in the women, and is to some extent combated by the frontal muscles, which, in contracting, make the patient frown and draw up the eyebrows. Ptosis may also be due to tubercular or syphilitic disease of the corpora quadrigemina, and the reason for

FIG. 6.



Medulla oblongata with the corpora quadrigemina. The numbers IV–XII indicate the superficial origin of the cranial nerves, while those (3–12) indicate their deep origin—*i. e.*, the position of their central nuclei ; 3 shows the deep origin of the oculo-motor nerve of one side.

this will be clear when the deep origin of the oculo-motor nerves from their nuclei is remembered. (Fig. 6.) Sometimes there will be associated with the ptosis internal squint due to paralysis of the abducens nerve (sixth), which arises from the nearby nucleus, and is connected with that of the oculo-motor (Fig. 7). (See also chapter on the Eye.)

If the condition is due to a serious congenital fault, we usually

find associated with it failure to elevate the eyeballs, and the failure is probably due to a nuclear defect. If due to gelsemium or conium, the symptoms of poisoning by those drugs will be present.

FIG. 7.

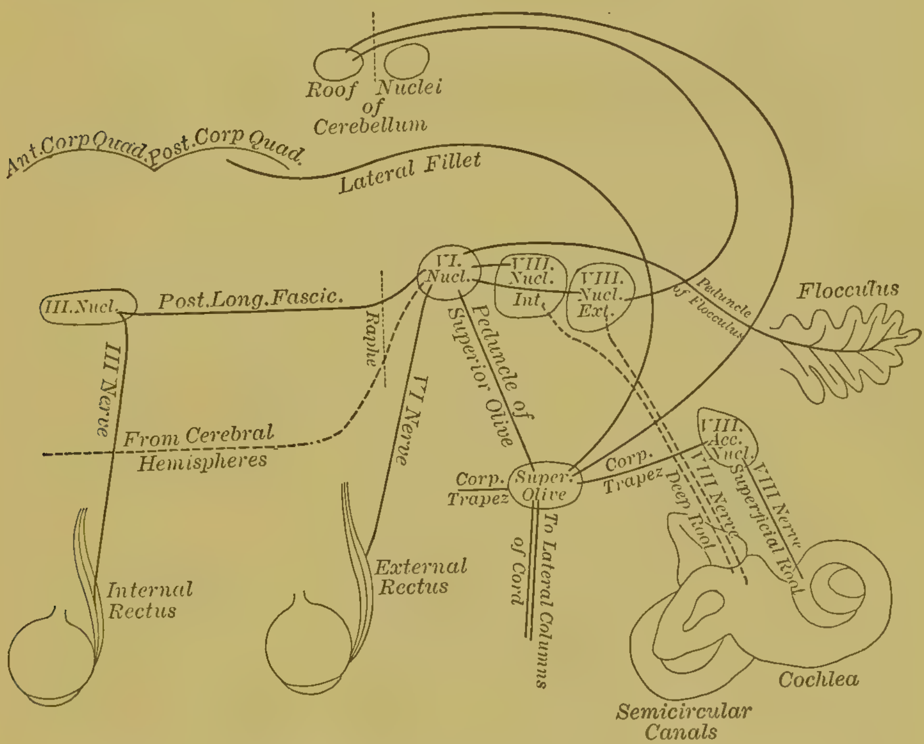


Diagram of the connections of the nucleus of the sixth nerve. (BRUCE.)

Ptosis, with hemiplegia of face and limbs on the opposite side of the body, associated it may be with hemianæsthesia, is due to a lesion in the crus cerebri, provided the two sets of paralyses occur simultaneously, otherwise they may be due to two separate lesions. (Hughlings Jackson.)

A very rare condition, of which there are but twenty-seven cases on record according to Darquier, is recurrent paralysis of the oculomotor nerve on one side. The attack begins with violent pain on one side of the head, nausea, and vomiting, and these symptoms are followed by ptosis, external strabismus, mydriasis, paralysis of accommodation, and crossed diplopia. It is seen most frequently in women, but may date from as early a period of life as eleven months. The attacks may last for a few weeks, and occur often or only after a lapse of many years.

Bilateral Facial Paralysis is a rare condition, and when it occurs can only be due to a bilateral lesion in the cerebrum, to acute bulbar

paralysis, to progressive bulbar paralysis, to a lesion in the pons just where the facial fibres decussate, to bilateral disease of the pons owing to disease of the basilar artery, syphilis at the base of the brain producing a tumor or inflammatory thickening, very rarely to bilateral inflammation of the mastoid foramina, resulting from cold or double otitis, from toxic multiple neuritis, but not from that toxic neuritis due to alcohol. Very rarely bilateral facial paralysis results from multiple neuritis in its diphtheritic form.

The development of bilateral facial paralysis due to a double cerebral cortical lesion never occurs without evidences of paralysis elsewhere in the body, such as monoplegia or hemiplegia.

The bilateral paralysis of the facial nerve in acute bulbar paralysis is characterized by its limitation, as a rule, to the neighborhood of the lips, by dysphagia, lingual paralysis, affected speech, paralysis of the ocular muscles, and a rapid pulse. This disease is very rare and depends for its existence upon an acute inflammation or myelitis of the medulla oblongata.

When due to progressive bulbar paralysis (glosso-labio-pharyngeal paralysis) the paralysis is confined chiefly to the lips, and is associated with alterations in the tongue (see chapter on the Tongue) and speech, with tremor of the tongue and stiffness of the lips. The mouth stands half-open, the lower lip is pendulous, and the patient's expression is that of a person about to burst into tears. The symptoms of glosso-labio-pharyngeal paralysis may, however, be exactly reproduced by diphtheritic paralysis, with this difference in prognosis: the first die and the second class get well.

In making a diagnosis of bulbar paralysis it should be remembered that another condition exists in rare instances in which no definite pathological changes can be found in the nuclei in the medulla oblongata, and yet many of the symptoms manifested by the patient are identical with those of glosso-labio-pharyngeal paralysis (true bulbar paralysis). This condition has been called "asthenic bulbar paralysis," and in it we find, as early symptoms, that the muscles of swallowing and of speech become easily tired on exertion, showing failure of the nuclei of the fifth nerve; that defects in articulation and speech are developed, indicating disorder of the nuclei of the ninth and tenth nerves; and clumsy movements of the tongue are present, which are a sign that the nuclei of the hypoglossal and twelfth pair are involved. These symptoms are practically identical with those of true bulbar paralysis. What are the

symptoms which by their presence in the true disease and their absence in asthenic bulbar paralysis aid us in separating the two affections? The answer to this question is that the drooling of saliva, the atrophy of the tongue, lips, and extremities, the fibrillary twitching of the affected muscles, and the loss of electrical irritability in these muscles, all of which symptoms belong to true degenerative bulbar paralysis, are not to be found in the asthenic form. There is, however, in the latter disease a condition rarely found in the degenerative form, namely, paralysis of the oculo-motor, the lower facial, and the inferior division of the fifth or trifacial nerve, causing dilated pupils, diplopia (which, however, is not accompanied by strabismus), and ptosis (from the oculo-motor failure), facial paralysis about the mouth (from facial nerve failure), and loss of expression about the eyebrows and forehead (due to facial and trifacial failure). Whether the diagnosis be true degenerative bulbar paralysis or the asthenic form just discussed, in both the prognosis is most unfavorable. Indeed, the asthenic form is often the more rapidly fatal of the two. In the latter the nuclei in the pons are probably always involved, but, as already stated, no pathological changes have been demonstrated in any of these nervous centres.

A very rare affection is oculo-facial paralysis, which is congenital or develops in childhood, and is chronic. There are paralysis of the ocular muscles and ptosis.

Facial Spasm. Spasm of the facial muscles may result from functional and organic disease, and occurs far more frequently in women than in men. The cause of the functional forms we do not understand, as they occur in neuropathic persons and about the climacteric period. Rarely the spasm arises from reflex irritation through the trifacial, resulting from a decayed tooth or a cause in the eye or in the skin. Habit-spasm arises from a trick learned by a child, or acquired as in taking snuff or in sniffling. The organic causes are many. Thus, there may be irritative lesion of the facial nerve-trunk or one in the cortical centre for the face, a tumor pressing on the nerve at its point of origin, or an aneurism of the vertebral artery. The spasm may be confined to one side or distributed over both sides, and is also seen in chorea, convulsive tic, blepharo-facial spasm, in tetanus, meningitis, and epilepsy. When due to chorea it nearly always is clonic or twitching, as it is also in convulsive tic, and habit-spasm, but in blepharo-facial spasm, tetanus, meningitis, and epilepsy it is generally rigid or tonic. In chorea

the spasm is most marked about the corner of the mouth and the eyebrow or eyelids. The movements of convulsive tic are exceedingly sudden, darting across the face and involving all the muscles supplied by the facial nerve. As a rule, it is unilateral. These spasmodic movements may be almost constant or appear in paroxysms, and rarely the muscles of the jaw, the neck, and the tongue are affected. The disease depends upon a disorder of the facial nerve, or its centres, which is not understood. The prognosis is bad so far as cure is concerned. Spasm of the levator palpebræ superioris muscle is sometimes seen as a symptom of exophthalmic goitre. It is called "Abadie's sign."

Spasmodic movements about the eyes such as have just been described are sometimes paralleled by what is called nictitating or clonic spasm, which is probably due to some undiscovered cause of reflex irritation. None of these spasms, however, are to be confused with what is called blepharo-facial spasm, a condition just described, in which there is ceaseless, rapid contraction of the orbicularis palpebrarum muscle. Generally in children there is also photophobia with a spasm of the eyelids which is often tonic in character and generally bilateral. This condition has associated with it what have been called "Graefe's signs," namely, the presence of spots near the supraorbital foramen or over the vertebræ, which when pressed on cause sudden relaxation of the spasm.

The development of facial spasmodic twitching accompanied by a sudden burst of explosive speech, repeating the last word heard or said by the child in conversation (called echolalia), or the sudden bursting out with some blasphemous or filthy word (called coprolalia), sometimes is seen in neurotic children, and is often associated with perversion of moral sense. It is called by Gilles de la Tourette "Maladies des tic convulsifs," but this is an unfortunate term, because it is apt to be confused with ordinary convulsive tic of children or adults. (See Electric Chorea and Myoclonus Multiplex in the chapter on the Hand and Arm.)

In tetanus the muscles of the jaw, the masseters, and temporals are first involved in the tonic contractions, and these are followed by rigidity of the muscles of the neck and body. Often the *risus sardonicus* is marked from the first, and the face soon looks very old owing to the muscular contractions.

In meningitis the characteristic symptoms which label the malady render facial spasm a comparatively unimportant symptom, and in

epilepsy the convulsive seizure soon makes easy the diagnosis of the cause of the facial spasm unless the epilepsy is limited in its character, when the history of the presence of an aura, or of unconconsciousness, or biting of the tongue may be discovered.

Spasm or contractures of the muscle of the face sometimes follow paralysis as recovery begins, and the contractures involve the formerly paralyzed muscles, whereas in paralysis in the limbs the contractures generally take place in the muscles which are not paralyzed. Sometimes these contractures in the face are permanent, and are due to incomplete restoration of the functions of the muscles affected. Care should be taken to remember that not very uncommonly contractures in the muscles of the face result from hysteria, and that they are often on the side opposite the facial paralysis if the latter exists.

Active spasm of the muscles of the face may follow exposure to cold and it sometimes follows the paralysis due to this cause.

Wry-neck consists in a drawing of the head to one side by the sterno-mastoid muscle in a state of spasm, and at the same time the head may be tilted to the back or front according to the accessory muscles which are involved in the spasm. It may be either congenital or acquired, and consists in a more or less constant clonic or tonic spasm of the sterno-mastoid muscle, which is, of course, involved on the side toward which the head is drawn, whereas the muscle on the opposite side is seen tense from being stretched by its opponent. Rarely the trapezius is the only muscle involved, in which case the head is drawn backward toward the diseased side, or, if these muscles are both involved, the head is tilted back until the patient looks up in the air. Pain in the muscles only occurs from fatigue. This tonic spasm affecting the head can be separated from that occurring in tetanus by the fact that in tetanus there is a general diffusion of the spasm to other muscles, although in that form of tetanus called "head or cephalic tetanus" the diagnosis is more difficult. This form of tetanus usually has with it the following diagnostic points: there is a history of infection, the character of the onset is sudden, there are trismus, difficult swallowing, respiratory disturbance, and facial paralysis with rare involvement of the ocular muscles. It is often increased by movement or the attempt to take food.

The Head.

The affections involving the head are those depending upon trophic changes in its size, and alterations in its position by reason of affections involving its muscles.

The spasmodic affections of the head, aside from those of the face, and wry-neck, are nodding spasm, tetanus, and chorea, although other spasms, such as epilepsy, often affect it.

Nodding spasm of the head, depending upon somewhat rhythmical contractions of the sterno-mastoid and trapezius muscles, is sometimes seen in half-fed or rickety children. It also occurs in hysterical women, and in men who are not hysterical. The nodding may be slow and infrequent, only coming on with excitement, or it may be practically constant. It always becomes worse when the patient is examined, and may be so rapid and forcible as to seem almost severe enough to shake the head off the shoulders. Often the muscles involved will be found on touching them to be very rigid.

The changes in shape are to a certain extent considered in that part of this chapter dealing with the symmetry and appearance of the face, but there still remain to be discussed the changes in the shape of the head as a whole. These occur in hydrocephalus, microcephalus, rickets, idiocy, and cretinism.

The head of hydrocephalus is greatly enlarged above the level of the ears, and this causes the face, already having a tendency to faulty development, to look small and wizened. The eyes seem somewhat bulging, the orbital plates are oblique, and the back of the head flattened. In microcephalus, on the other hand, the head is small and often narrow. Technically, this term is applied to idiots whose heads are less than seventeen inches in circumference. Nearly always the head of an idiot is abnormally formed. The cretinoid head is large, heavy, and massive.

When a young child has unusually prominent parietal and frontal bones, which seem bulging, and there is a general resemblance of the shape of the skull to that of hydrocephalus, we suspect the presence of rickets. As a rule, the forehead is broad and high, and the top of the head flat. Sometimes in such a child we find, in addition to these changes from the normal, spots of thinned bone in the occipital and parietal regions. These may be also somewhat softened, and this condition, called "cranio-tabes," is usually a sign of rickets which exists in association with infantile syphilis. Rickets is seen

nearly twice as often in boys as in girls, and there is usually to be found deficient development of the bones everywhere, particularly in the ribs and legs.

Excessive sweating of the head, producing a wet pillow, is often an indication of rickets when it occurs in a child.

Retraction of the head in children is an indication in many cases of serious brain disease, and commonly arises from a basal meningitis, probably as the result of an effusion into the ventricles. It is to be remembered that some of these cases recover, though such a result is rare. Again, we should not forget that caries of the cervical vertebræ may cause this position, or that tender and enlarged glands in the neck may so result. Sometimes, too, it occurs after falls without there being any indication of meningeal irritation. Rarely in neurotic babies retraction of the head, as a temporary symptom, accompanies attacks of indigestion.

CHAPTER II.

THE HAND AND ARM.

The general appearance of the hands and arms—The shape of the hand in disease—Spasms of the fingers—Tremors of the hand—Paralysis of the hand and arms.

THE appearance of the hand and arm often gives us valuable hints in the diagnosis of disease, chiefly by reason of variation in its shape, manner of movement, and general consistency; but as all these conditions vary widely in normal individuals, we can only regard distinct and well-marked alterations from the normal type as indicative of a definite disease. We can, however, often gather general information as to the patient from the hands, particularly as to his occupation; thus we see the smooth, soft hand of the professional man or clerk, the horny hand of the laborer, the blackened nails and skin of the machinist, or the blue-black dottings of the hand of the miner; and Hirt asserts that atrophy of the antithenar eminence often ensues in cabinet-makers, perhaps from the excessive use of the plane. Even when no pathological condition exists we are wont to regard the heavy and somewhat thick and clumsy hand as an evidence of a phlegmatic temperament, and the thin, wiry dexterous hand as indicative of the nervous temperament. Similarly, we recognize as the hand of the strumous that one in which the fingers are slender between the joints and the joints themselves thick and clumsy, or, again, in persons with tubercular tendencies, we see a slender, delicate hand easily compressed and somewhat effeminate in type. Very commonly, too, in children who have developed heart disease in early life, the hand becomes square-looking, and the fingers are club-shaped through thickening at the tips. A similar clubbing also manifests itself in many cases of emphysema and chronic phthisis in adults.

From the appearance of the nails we can often gain important information; thus, whenever the color of the blood in the capillaries under the nails is dusky we know that a deficient pulmonary function exists or that the circulation is impaired, it may be from feebleness or from cold. In anæmia the nails are often very pale, and

Stephen Mackenzie has asserted that if pressure on the tip of the finger completely empties the capillaries under the nail so that the appearance is pale the red corpuscles are present in only half the usual number.

White spots in the nail may be due to injury of the matrix by picking at the base of the nail, or be due to acute fevers producing trophic changes.

When the nails are striated and in longitudinal ridges the patient is often of the gouty diathesis, while transverse ridges may indicate arrest of nail-growth through local injury to the matrix or the impairment of the nutrition as the result of some severe systemic shock, as a severe surgical operation or prolonged illness. Sometimes these marks result from a severe attack of gout, and Fothergill tells us that it took about seven months for such a mark to grow out of his nails. Ordinarily, this mark will be found about half-way up the nail three months after the attack. In hemiplegia or acute infantile palsy the growth of the nail of the paralyzed part is generally arrested, as can be determined by staining it and watching to see if the stained part gradually moves away from the base. When the nails are distorted and thickened the cause may be local injury or peripheral neuritis, or any condition of the nervous system resulting in decided trophic influences, as in syringomyelia.

FIG. 8.

Dactylitis syphilitica in the infant. (TAYLOR.)

Hypertrophy of the nails so that they are abnormally elongated is usually associated with thickening and the development of great fragility. The nail may even be spirally twisted (onychogyrophosis),

or, if very wide, may cut into the skin and produce paronychia. These conditions may result from skin lesions, such as eczema, or lichen ruber, at or near the matrix, or be due to syphilis, and Vogl asserts that mere thickening may arise from severe fevers. They may also be seen in cases of Raynaud's disease, or in sclerodactyle, and in pulmonary osteo-arthropathy.

Atrophy of the nails may apparently arise from identical causes with those which produce hypertrophy, and Kaposi has seen the nails soft and membrane-like, with abscesses under them, from psoriasis of the fingers. A diagnostic indication given us by the fingers is seen in dactylitis due to syphilis (Fig. 8), or in other cases this is replaced by an eruption on the skin of the hand characteristic of this disease; another is seen in the ulcers at the bases of the finger-nails, with ecchymotic spots on the skin, produced by the chloral-habit; and still another is the sores seen at the bases of the finger-nails in persons who handle irritating drugs, such as elaterium.

Congested veins on the hand may indicate obstruction to the venous circulation of the arm, or general lack of vascular tone and a feeble heart.

When the hand is cold and clammy the condition may be due to bromidrosis, or a local disturbance in innervation of the sweat-glands. It is often seen in cases of so-called spinal irritation and nervous exhaustion. Excessive sweating of the hand is also often seen in cases of progressive muscular atrophy.

There are two sets of movements associated with the muscles of the wrist and hand which possess grave prognostic and diagnostic importance. The first of these is twitching of the muscles of the forearm (subsultus tendinum). It indicates severe exhausting disease. The second is picking at the bedclothes. The description of the grave import of this dangerous symptom, "picking at the bedclothes," or carphologia, is given by Shakespeare in his description of the death of Falstaff: "After I saw him fumble with the sheets, and play with flowers, and smile upon his fingers' ends, I knew that there was but one way; for his nose was as sharp as a pen." And again, Hippocrates has well emphasized the gravity of this symptom, for he says: "In acute fevers, in peripneumonias, in pleuritis, and in headaches, the hands are moved to and fro about the face seeking in the void, as if gathering bits of straw, picking at the coverings, or detaching objects from the walls of the room, constituting so many signs of a fatal termination."

but often pallid, particularly in the puffy, œdematous area on the back of the hand. The presence of intense local inflammation, the

FIG. 14.

A Röntgen ray picture showing the condition of the bones of the hand in a case of chronic rheumatoid arthritis. It will be seen that the peculiar outlines of the proximal phalanges are due to their positions—as lesions we may note anchylosis of the metacarpal of the middle and ring fingers with the os magnum and unciform deposits in the heads of the phalanges and dislocations. (From the *Medical Chronicle*, April, 1896.)

history of sudden onset, and the intense pain on movement readily separate rheumatism from gout and arthritis deformans, and leave it to be separated from sprain, septic arthritis, and deep-seated inflammation of the hand proper. The first is excluded by the history, the second by the history and general lack of evidence of gonorrhœa or sepsis or purpura, and the third by the lack of accompanying general systemic disturbance and the absence of a history of traumatism or infection.

The nervous disturbances which change the appearance of the hands are very numerous.

Angioneurotic œdema is not peculiar to the hand, although fre-

quently involving this part of the body. It consists of a swelling varying in size from a dime to a silver dollar, which is not œdematous in the sense that it can be pitted on pressure. This swelling, which may be multiple, red in color, or pale and waxy in appearance, lasts but a few hours or days, disappears, and often speedily returns. Somewhat allied to angioneurotic œdema is that condition of the hand (or toes) characterized by a white and waxy or slate color of the fingers, associated with coldness, swelling and mottling of the skin, termed "Raynaud's disease." Often this is a passing condition, but in its severe forms there is finally developed dry gangrene of the fingers involved. The conditions of the hand resembling it, from which it must be separated, are senile gangrene, in which the advanced age of the patient and the presence of diseased and thickened bloodvessels will enable us to decide on the latter as the cause; frost-bite, in which the history of exposure will be of value, although exposure to cold often precipitates an attack of Raynaud's disease; ergotism, which can be discovered by the history of the patient having taken food for a long time which may have contained bad rye; leprosy, which will probably be seen more marked in other parts, and in the patches of which can be found the leprous bacillus; and alcoholic neuritis, of which we shall speak later (see chapter on the Skin). In that state known as Morvan's disease or "pain-anæsthesia with whitlow," there is a slowly progressive loss of power in the hand with atrophy and ulcers about the bases of the nails. Sometimes the terminal phalanges undergo necrosis, and enlargement of the fingers, through swelling, may be very marked. It is probable that this condition represents two separate lesions, namely, neuritis and syringomyelia, and it is an exceedingly rare disease.

In addition to these trophic changes in the hand we have the so-called "spade-like" hand seen in myxœdema, acromegaly, and the pulmonary osteo-arthropathy of Marie. In myxœdema the deformity depends upon the alterations in the subcutaneous tissues, rather than on changes in the bones, so that the hand is swollen or boggy-looking, but does not pit on pressure as in true œdema. In acromegaly the enlargement is chiefly osseous, as it is also in pulmonary osteo-arthropathy, the formation being on a gigantic scale. In the latter disease, however, the hands and feet are alone affected, and the enlargement is not symmetrical. Further, this condition is nearly always associated with changes in the lungs, such as emphysema, tumors, and old bronchial troubles. The hands are not only

greatly enlarged, but deformed, so that a side-view of the finger-tips reminds one of the shape of a parrot's beak, the nail being turned over the end of the finger. This is even more marked in the thumb.

Alterations in the contour of the hand are, however, far more frequently produced by atrophic processes than by those which result in hypertrophy. They arise in cases of paralysis not only from wasting of the muscular tissues, so that hollows or sunken places occur, but also from the distortions caused by the contractions of healthy muscles, which, having no opposition as in health, speedily draw the bones of the hand into abnormal positions. In other cases the diseased muscular fibres may be spasmodically contracted, overcoming the resistance of the healthy muscles.

The wasting of the hand seen in old age, particularly in women, and in advanced phthisis, diabetes mellitus, and other conditions in which the tissues of the body in general lose their plumpness, is so universally distributed that a diagnosis of wasting from old age is not difficult. On the other hand, the wasting due to nervous lesions is generally not universal, but limited to single muscles or groups of muscles, the remaining portion of the hand having its normal appearance, or being only indirectly influenced.

FIG. 15.

Claw-hand. (GRAY.)

Under the name of "claw-hand," or "*main-en-griffe*," we find a deformity of the hand which is in itself very characteristic, although indicative of several causes which all operate in an identical manner. The back of the hand loses its normal convexity and becomes somewhat concave, the tendons on the extensor surface stand out in ridges, the proximal phalanges are drawn backward toward the wrist, while the second and third phalanges are drawn toward the

palm of the hand (Fig. 15). Sometimes, however, the tips of the fingers are drawn toward the back of the hand. This deformity results from atrophy and paralyses of the interossei muscles and lumbricales, which are supplied by the median and ulnar nerves. The extensor communis digitorum and flexor digitorum produce a dorsal flexion of the first phalanges and a complete palmar flexion of the second and third phalanges. A certain amount of immobility is also caused by the fact that flexion of the hand is impossible in the fingers and almost lost at the wrist.

The claw-hand having been recognized, it remains to be decided what are its causes. It may be due to disease of the peripheral nerves (the ulnar and median), of the cells in the spinal cord, and of the cells in cerebral cortex in the hand-area.

Taking up for consideration paralysis of the median and ulnar nerve as a cause of claw-hand, we find that the most common cause is a neuritis produced by some mechanical injury resulting from an accident, or from the following of some occupation in which, for example, the artisan presses his elbow constantly on some hard surface. The deformity may be, therefore, either unilateral or bilateral (generally the former), and there will be evidences of local injury, or a history which will indicate that the lesion is peripheral. Further than this, there will nearly always be found, in ulnar and median injury, sensory as well as motor paralysis; and Hirt asserts the remarkable fact that the claw-hand may develop in cases in which sensory disturbances are the only evidence of median and ulnar difficulty; in other words, before motility is lost through paralysis. (See chapter on the Skin, Anæsthesia of the Skin.) Toxic neuritis very rarely, if ever, causes claw-hand, as the musculo-spiral nerve is more commonly affected in these cases and the extensors become paralyzed.

There are several spinal causes of claw-hand, the most important of them being progressive muscular atrophy, that disease in which there are atrophy and abnormal change in the anterior horns of the gray matter of the spinal cord, particularly in the cervical region. (Fig. 16.) It will be remembered, too, that the anterior nerve-roots and motor nerves become involved in this process. As a result of these changes, we have developed loss of power in the hand and arm followed by the development of a claw-hand from wasting of the same muscles, as already described, the disease-process being generally bilateral, but affecting the right hand and arm more than the left as a rule. As progressive muscular atrophy often

in that very rare condition called Morvan's disease, but it has not the characteristic appearance of *main-en-griffe,* there being a slow symmetrical wasting of the muscles with a drawing of the fingers into flexion. There are also analgesia and painless whitlows. It usually occurs in young or middle-aged males. Morvan's disease of the fingers, as already stated, may arise from a syringomyelia and neuritis, or neuritis alone.

Another spinal lesion producing great alterations in the appearance of the hand and arm, through wasting of the thenar and antithenar and interossei and the muscles of the arm, is amyotrophic lateral sclerosis. Here again the hand often shows the first manifestations of the disease in the loss of power of which the patient complains. The early symptoms of amyotrophic lateral sclerosis may closely resemble those of progressive muscular atrophy in the loss of power in the thumb muscles, but in this disease the reflexes are markedly increased in the affected muscles, whereas in progressive muscular atrophy they are lost, although fibrillary muscular twitchings may be caused by tapping.

Again, the patient is usually manifesting some of the symptoms of lateral sclerosis when he comes before the physician, such as weariness, stiffness, and loss of power in the legs (see chapter on Legs, Paraplegia). There are also exaggerated knee-jerks and ankle-clonus, and wrist-jerk is marked.

Wasting of the muscles of the hand, causing distortion, may also be due to syringomyelia, but generally there will be with this loss of power and disturbance of sensation, such as anæsthesia. Often in syringomyelia there will be developed an arthropathy of the arms such as is seen in the legs in tabes.

Wasting of the hand, with flexion and rigidity and sometimes contractures, is seen rarely in advanced paralysis agitans in place of the characteristic tremor.

In the "cerebral palsy of children," sometimes called "spastic infantile hemiplegia," the hand may be flexed on the forearm, and the forearm on the arm, the thumb drawn into the palm of the hand and the fingers flexed as in Fig. 20. These deformities are not necessarily confined to one arm alone, but are sometimes bilateral. A peculiarity of these cases is that the muscles waste very slightly, and do not develop the reactions of degeneration, so that the case separates itself from poliomyelitis. The fingers in the cerebral palsy of children can often be placed in curious positions with ease, and, if the limb

be suddenly flexed, a lock-like sensation will be imparted to the physician's hand. Convulsive seizures of an epileptiform type are very frequent. Cohn asserts that there are on record eight cases in which intention-tremor has taken the place of the spastic rigidity just described, and he reports a ninth. Similar lesions may follow infantile cerebral hemorrhage, thrombosis, or embolism. (Fig. 20.)

FIG. 20.

Right hemiplegia, with contractures and retarded growth of arm. Onset of disease at eight years of age, following typho-malarial fever. (SACHS.)

Again, in persons who have had apoplexy it is not uncommon as time goes on for the temporary spasm seen in the muscles of the hand and arm to be replaced by permanent contractions resulting in deformity. These contractions, if they occur early, are an evidence of irritation of the pyramidal tract or the fibres just behind the knee of the internal capsule, and are of serious import if they come on early, as they show the extension of inflammatory processes. When they come on later they show that a degenerative process is descending the pyramidal tracts. Wasting finally comes on. (For further discussion of the significance of paralysis in the arm and hand, see succeeding pages and chapter on Hemiplegia.)

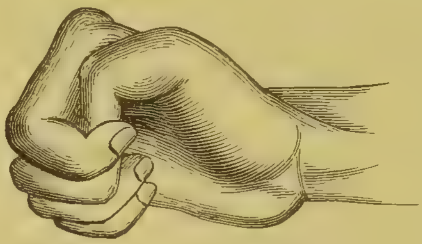
A very important point always to be remembered in examining contractures of the hand and arm, or of the lower limbs, is the fact that they often are due to hysteria, in which case the history is that they set in suddenly, and they are generally accompanied by other hysterical manifestations, which can be discovered if sought for. As a rule, the muscles do not waste or develop degenerative reactions, but rarely such wasting may occur. (Figs. 21 and 22.) Care must be taken in giving a prognosis for cases of hysterical contracture, since organic lesions sometimes supervene. Charcot states that if the contractures persist when the patient is under anæsthesia, and the muscles are atrophied, organic disease exists. It is important to remember this, for these contractions may be practically permanent when once induced, and as injuries may produce a true organic or a false

hysterical contracture, much medico-legal interest centres about this differential diagnosis. Closely allied to these cases are those of hysterical contracture, in which after grasping an object the patient cannot let go until the muscles are stroked. Putting an Esmarch bandage on such a forearm will usually produce the spasm.

FIG. 21.

Hysterical atrophy of the hand with flexion of the last two phalanges into the palm, particularly the last phalanges of the index and middle fingers. (GILLES DE LA TOURETTE.)

FIG. 22.



Same hand in another view.

When in the course of an acute illness in a child the fingers are drawn down into the palm of the hand, with the tips touching the palm and the thumb turned in beneath them with its tip pressing the palm, the patient may have meningeal congestion or inflammation, or hydrocephalus, and a general convulsion may be imminent.

When the fingers are bent toward the palm, but the tips extended and the thumb turned in ("the accoucheur's hand"), the position is typical of tetany, but in this condition the rest of the body will give evidence of involvement. The nervous irritability in this condition is greatly increased, and pressure on a large bloodvessel or nerve-trunk will often produce the spasm. Curiously enough, gastric dilatation or thyroid wasting will often be found with tetany. In other cases it appears to be due to profound debility, as after prolonged nursing. Care must be taken to separate the so-called carpopedal spasm of rickety, hydrocephaloid children from true tetany, in which the body is usually involved, and from spastic paralysis due to infantile cerebral palsy.

Spastic rigidity of the arms is often one of the earliest signs of chronic hydrocephalus, even before the skull begins to enlarge, and convulsions may be present from time to time. In congenital spastic rigidity due to sclerosis or defective development of the cortex cerebri the spastic condition is usually confined to the legs. (See chapter on Legs and Feet.)

Spasm of the fingers of a rigid type on attempting to make certain movements is also seen as the result of excessive use of the part involved, and occurs in seamstresses, cigarette-rollers, cigar-rollers, typewriters (rarely), telegraphers, milkers (rarely), persons who use a pen to excess, and in piano, flute, clarionet, and violin players, or in persons engaged in any occupation requiring constant and comparatively minute and well co-ordinated effort. It seems to be more common in men than women by a large proportion (39 to 4).

Sometimes paralysis, tremor, or vasomotor disturbances take the place of occupation-spasm.

The spasm resulting from occupation must be separated from that sometimes seen in the hand in post-hemiplegic chorea, progressive muscular atrophy, the various forms of toxic peripheral neuritis, and that due to irritative cerebral foci, such as tumors of the brain. The history nearly always clears up the diagnosis. Spasm of the muscles of the hand and arm, rhythmical or otherwise, may also be due to hysteria, and may resemble, when due to this cause, true tetany (not tetanus).

Choreic movements are seen chiefly in children as a manifestation of chorea minor. They are usually seen in rheumatic and neurotic children, and heart-murmurs are heard in these cases. The first evidence of spasm may be developed in the hand, and be limited to that member in rare cases, and the hand often drops things that are placed in it. The hand itself is rarely involved alone, and the muscles of the arm toss the entire arm and hand from spot to spot with a fidgety, jerking movement which is very characteristic. A form of chorea minor, usually limited to the arm, is called paralytic chorea. It comes on suddenly, and is characterized by loss of power with a few feeble twitches. It affects only children.

Sometimes choreic movements come on in the latter half of life, often preceded by emotional disturbances. These movements are not true chorea. They are often called senile chorea.

In some cases of adult chorea the patient tends to become maniacal, particularly toward night. Such cases usually occur in women, and the prognosis as to life is bad. There is often in these cases great mental heaviness.

Several other affections which somewhat resemble true chorea are sometimes met with, but all of them lack, with one exception, the peculiarity of its movements. One of these is what has been called habit-chorea, or, more correctly, habit-spasm, in which condition the

patient acquires a nervous trick of jerking a muscle or a set of muscles. Unlike true chorea, it is more frequently seen in adults than children. Its limitation, as a rule, to a single set of muscles and the history of the case usually separate it from chorea minor, and the muscular movements consist in sudden twitchings rather than jerking, irregular muscular movements.

In paramyoclonus multiplex the disease, as the nerve implies, usually involves symmetrical parts, the contractions of the muscles appear in paroxysms, and the muscles involved are usually the biceps, deltoid, and triceps in the arms, and the quadriceps femoris and calf muscles of the lower limbs. Myoclonus multiplex is a disease of adult life, and chorea is usually seen in childhood. Sometimes the muscles in myoclonus are exceedingly irritable.

Under the name of electric chorea, or "Dubini's disease," Dubini described a disease, affecting both sexes and all ages, in which sudden shock-like contractions of the muscles take place, as if they were being stimulated by a slowing interrupted faradic current. The disease usually begins in the upper extremities and gradually involves the rest of the body, and progressively passes to a fatal issue. This is a very rare disease, and the sudden contraction of the muscles in tonic spasm separates it from chorea.

Still another form of electric chorea is that of Bergeron, which is probably identical with what has been called hysterical chorea. Here, again, the shock-like muscular contractions are manifested chiefly about the shoulders. The patient is usually a female, and has the stigmata, sensory and otherwise, of hysteria (see chapters on Skin, Eye, and Feet and Legs).

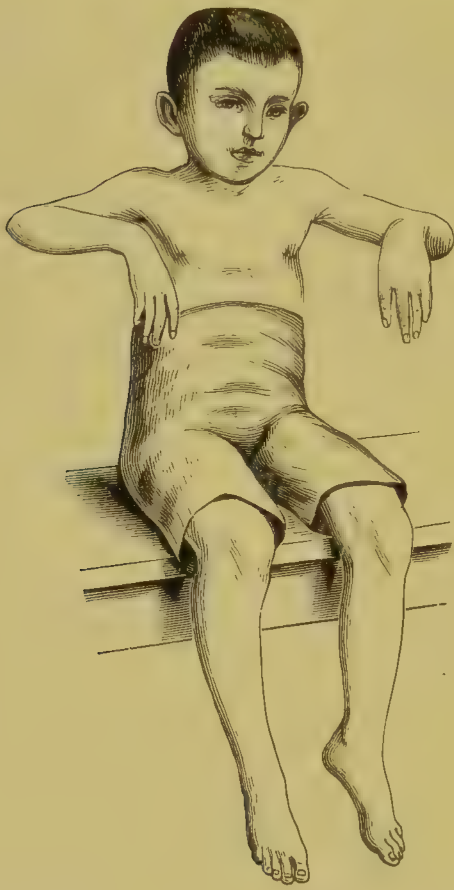
Again, the physician may meet, exceedingly rarely (almost never, in the United States), with a condition called convulsive tic or palmus, which has also been called "the jumpers," in which the movements are not in the slightest like true chorea, but are sudden muscular movements, usually imitative of the act of some other person or animal. It is often associated with echolalia—that is, repeated or echoed speech—or coprolalia or filthy speech.

Finally, another very rare disease is that known as Huntington's or hereditary chorea, a condition in which the twitching usually begins in the face and extends to the arms and legs. This ailment is hereditary, rarely begins before thirty years of age, is accompanied by progressive mental deteriorations, a tendency to melancholia, and may last ten or twenty years.

Mercurial poisoning producing tremor may cause so coarse a movement in advanced cases that the case may be thought choreic. (For tremors, see latter part of this chapter.)

In "Thomsen's disease" the hand is placed in tonic spasm as soon as voluntary movement is attempted. Closely resembling Thomsen's disease, or myotonia congenita, is what is called paramyotonia, which exists in three forms: First, a patient suffering from paralysis agitans on attempting to move is seized with rigidity of the muscles, which holds him fixed; second, a patient is suffering from ataxia and muscular weakness, and is seized with an attack of muscular rigidity;

FIG. 23.



Boy with multiple neuritis, with double wrist-drop and slight foot-drop. (SACHS.)

and, third, a patient may have the muscular fixation occurring just as it does in Thomsen's disease, save that it is produced by cold or exposure, and not by intention-movement, and may last for hours. This is called paramyotonia congenita. (See also Athetosis.)

The position of the hand may be very various. Thus the hand

may drop edgewise from the radius toward the ulna from paralysis of the extensors on the radial side of the forearm resulting from neuritis or acute infantile poliomyelitis, while marked drop-wrist may occur from paralysis of the extensors in chronic lead-poisoning, or in any form of neuritis, toxic or otherwise, involving the nerve-supply of these muscles (musculo-spiral nerve). (Fig. 23.) Wrist-drop may also be developed by pressure upon the musculo-spiral nerve, as in crutch-palsy. If the wrist-drop is bilateral, it may be due to toxic neuritis; but if unilateral, it is probably but not positively due to pressure-paralysis from sleeping with the head resting on that arm, or from pressure by a crutch. Very rarely unilateral wrist-drop is seen in lead-poisoning. When lead is the cause the supinator longus usually escapes, as does also the short extensor of the thumb, so that the forearm can be flexed and the thumb extended. Pain is rarely present in pressure or lead wrist-drop, but is present in wrist-drop due to alcoholic and other forms of toxic neuritis. Often, too, in these cases the flexors are considerably involved (see part of this chapter on Brachial Monoplegia).

Tremors of the Hand and Arm. The movements of the hand should always be carefully watched in cases of suspected nervous disease. The most common alteration will be found to be tremor, which may indicate paralysis agitans, general paresis, chronic mercurial, plumbic, or alcoholic poisoning, hysteria, senility, Graves's disease, and disseminated sclerosis. Sometimes a tremor may be found in naturally nervous women who are drinkers of tea to excess.

In paralysis agitans the whole hand is involved, and generally both hands are equally affected. The tremor is rhythmical and fine or minute in character at first, but later may be quite coarse. It is a slow tremor of about five vibrations per second, which is more or less constant, and worse when attention is called to it, but it is not greatly increased, and, perhaps, is even decreased, by a voluntary act, such as an attempt to raise a glass of water. Very rarely, however, the reverse holds true, and the tremor is increased by voluntary effort. The fingers are generally semi-extended and the thumb is adducted, so that it constantly rubs the index finger with its pulp, as if it were attempting to rub off the skin of that member. Frequently there are pain and aching of the extensor muscles of the forearm and wrist from the constant exertion. (See chapter on Feet and Legs, part on Gait.)

The tremors of disseminated sclerosis are also slow, but coarse in

character. They are not constant, but are developed upon intentional movement, and have a greater amplitude than those of Parkinson's disease (paralysis agitans). Often threading a needle will be possible for a person with this disease, because it is a short act, while lifting a glass of water will be impossible. The other symptoms of disseminated sclerosis beside intention-tremor are staccato or scanning speech, exaggerated knee-jerks, ankle-clonus, jaw-jerk and wrist-jerk, disordered speech, nystagmus, muscular weakness, with rigidity and paralysis of groups of muscles, causing monoplegia, paraplegia, or paralysis of the cranial muscles.

The tremor of mercurial, plumbic, and alcoholic poisoning resembles that of paralysis agitans, save that it is more rapid, reaching nine or ten vibrations per second, and in the case of alcoholic tremor is decreased by a large drink of liquor, while those due to lead and mercury may be rapidly relieved by potassium iodide. Further than this, the tremor of alcoholism is generally worse in the morning.

A point of some importance in plumbic neuritis producing tremor and wrist-drop is the fact that painful sensations are rarely present; in arsenical neuritis, on the other hand, they are often the most prominent symptom, even preceding the motor disturbance. In mercurial neuritis, on the other hand, tremor precedes all evidence of loss of power, and, finally, may become so coarse as to resemble chorea. The tremor of general paresis is also rapid, eight or nine per second, and is a very fine tremor, which may be felt only when the arm is extended and the finger rested on the hand of the physician. In other words, the tremor of the hand of general paresis is generally not a predominant symptom, but is elicited when the muscles are put upon a strain. In regard to the fineness of the tremor of general paresis, it should be remembered that it closely resembles that of Basedow's or Graves's disease (exophthalmic goitre) (eight or nine per second), since the tremor of this condition is not only equally fine, but generally unseen except when the arm is extended and tips of the fingers rested upon the fingers of the doctor. This tremor has been called the "railroad bridge tremor," because of its fineness and vibratory character. The individual fingers do not separately tremble in Graves's disease.

In post-hemiplegic tremor the trouble is unilateral, and there is a history of cerebral injury and paralysis is present.

Tremor of a very marked character may be due to hysteria, and arises most frequently in those who have been exposed to shocks or

accidents. The tremors may occur constantly or only with intention-movements, or be increased in amplitude but not in rhythm on movement. The latter form is known as the "type Rendu," and has a rhythm of seven to nine per second, while the slower hysterical tremor may be four or five per second.

Beyond the state of tremor should be recalled the movements of chorea, which may be limited to one arm or hand, and which in their milder forms may be confused with the pronounced movements produced by effort in disseminated sclerosis. The latter are often very arhythmical, and so the choreic movement the more closely resembles them; but those of sclerosis are purposive, while those of chorea are not, since the movement contemplated in chorea is opposed by a contradictory contraction.

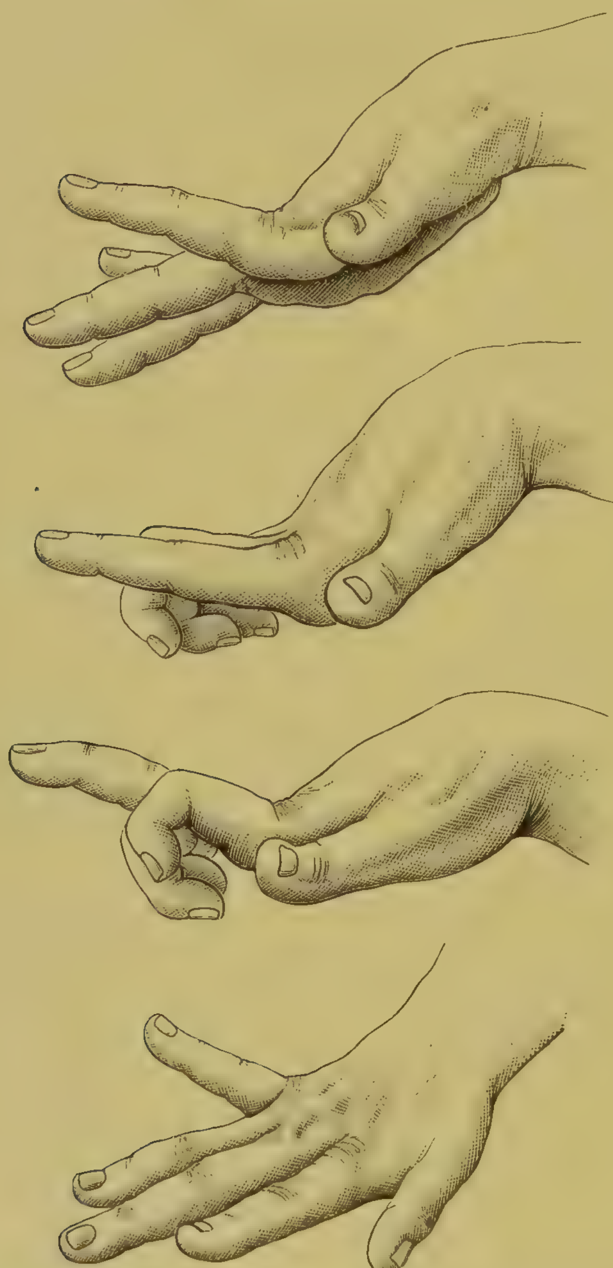
Movements of the Hand and Arm. Aside from the movements of tremor, careful note should be made of the movements of the hand as a whole, and of the co-ordination of its fingers and of the arm governing it. Thus, trembling contractions of the extensor tendons (subsultus tendinum) is a sign of grave and advanced forms of typhoid fever, and picking at the bedclothes (carphologia) is of still graver import (see beginning of this chapter). Inability to write, to play musical instruments requiring the use of the fingers, or to sew, may indicate the rare form of locomotor ataxia involving the upper extremities, so that if the patient is asked to close his eyes and feed himself the fork or spoon misses his mouth through lack of co-ordination, although loss of power may not be present.

Sometimes in locomotor ataxia as the disease becomes advanced paroxysmal twitching of the fingers may come on, or involuntary movements of the fingers occur in association with voluntary movements elsewhere.

In locomotor and Friedreich's ataxia also the movements of the hand are often lacking in co-ordination. The hand may be advanced past the object which the patient desires to grasp or else falls short of it. On endeavoring to pick up an object the fingers are spread over it like a widespread claw. Generally these ataxic symptoms will be more marked in the other parts of the body and be bilateral, but Ormerod has reported an instance in which only one hand (the left) was involved. This faulty movement of the hand may, however, be due to the fact that the ocular muscles are affected, and the "erroneous projection" due to this cause leads the patient to pass beyond the object reached for.

When fibrillary twitchings of the muscles are present and tapping the muscles produces idiopathic muscular contraction progressive muscular atrophy may be present.

FIG. 24.



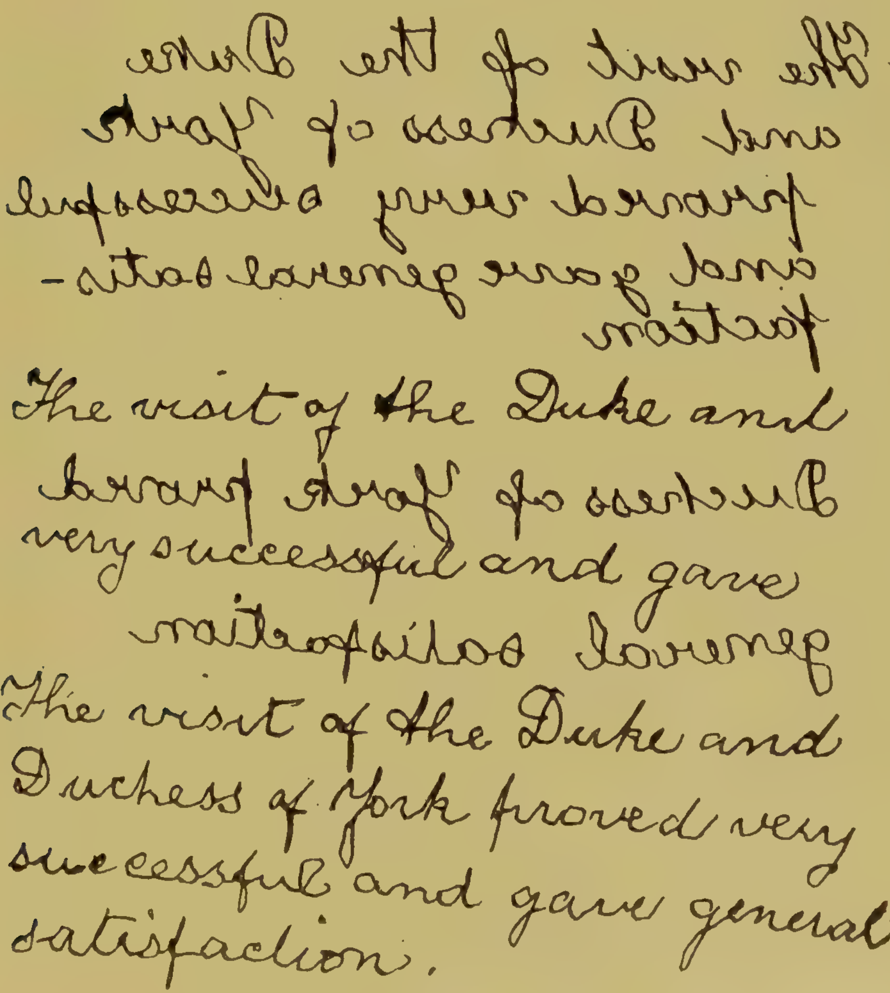
Examples of the position of the fingers in the movements of athetosis. (STRÜMPELL.)

Sometimes, as the result of infantile cerebral paralysis or from lesions developing in later life, the muscles of the hand are affected

by a slow, constant movement, so that the fingers assume curious constrained and unusual postures, being moved into extreme or forced extension, flexion or pronation, or supination. (Fig. 24.) This condition is called athetosis, and is separable from chorea in that the movements are slower and limited to the fingers and wrists, the arm escaping.

In this connection mention should be made of "mirror-writing," a curious condition in which the patient writes from right to left instead of left to right. It occurs in some cases of mental feebleness, hereditary or acquired, and rarely in hysteria. "Mirror-writing"

FIG. 25.



may also be present in cases of cerebral paralysis. The above example of this, taken from the case reported by Clapham, illustrates the character of the handwriting. The patient, a girl of twenty-

four years, could write all three ways, but mirror-writing was easiest to her. (Fig. 25.)

Very rarely athetoid movements of the fingers occur in advanced spinal tabes, probably as the result of a related lesion, and not from tabes itself.

Brachial Monoplegia. Absolute loss of power in the hand and arm without the necessary development of subsequent deformity results from cerebral and peripheral lesions, as a rule, being rarely spinal in origin, and is called brachial monoplegia. The causes of this loss of power when its cause is cerebral may be various. Thus, the lesion may be cortical or subcortical; that is, in the surface of the brain or in the internal capsule, or between the cortex and the capsule in the corona radiata. As a rule, however, monoplegia is cortical in origin, for below the cortex the motor fibres run so closely together that only a very small lesion can involve one without involving all, and so producing a hemiplegia. These cortical lesions when they do occur are generally, but not always, associated with convulsive seizure in the paralyzed limb, and Seguin has called this convulsion the "signal-symptom" indicating a cortical lesion. Brachial monoplegia not due to hysteria or neuritis, preceded and accompanied by a convulsion and loss of consciousness, and lacking in signs of involvement of lower nervous centres, is, therefore, cortical, and is generally due to the formation of a clot in the hand and arm centre resulting from injury or from the ordinary vascular causes of apoplexy. In other cases it is due to cerebral embolism or thrombosis, or to the growth of some neoplasm, specific or otherwise, or to a localized meningitis.

The probability of the lesion being an embolism or thrombosis is decreased by the recollection of the fact that the cortex is so well supplied by vessels from the pia mater that paralysis of a centre from lack of blood-supply from such a cause is rare, unless the lesion is subcortical, or, in other words, not deep enough to involve fibres from other centres as they approach each other, and yet sufficiently deep to prevent the nutrient blood-supply from the pia mater as just mentioned. Aside from the discovery of a condition of the internal organs, such as cardiac valvular disease or sepsis, which might cause embolism, the diagnosis between paralysis from hemorrhage and embolism is practically impossible, and this is also true of the paralysis due to thrombosis, except that in cases of thrombosis we often find the presence of general endarteritis, and the paralysis of thrombosis

may be slow and gradual in its onset. If the paralysis rapidly spreads, the lesion is probably due to a hemorrhage.

The history of there having been some sudden cause for an increase in arterial tension, as by muscular effort, and the presence of atheromatous vessels aid us in deciding as to the probability of the lesion being due to a hemorrhage, and the history of the sudden onset, coupled with the symptoms named, makes the diagnosis clear in a certain proportion of cases.

Neoplasms or tumors of the brain producing monoplegia are gradual in their development, accompanied generally by headache, by changes in the optic disks, and sometimes by mental disturbances or pressure-symptoms. A specific history pointing to the formation of a syphilitic tumor is of value in the diagnosis. (See chapter on Headache.)

If brachial monoplegia results from a lesion in the internal capsule, the lesion must be very limited, or, in other words, only large enough to cut off the hand and arm fibres. Tumors and lesions from traumatisms in this area are very rare, and hemorrhages, which frequently cause paralysis by affecting this area, are generally profuse enough to cause hemiplegia—that is, injury of the motor fibres supplying the leg muscles as well. Sometimes, however, sudden inflammatory processes are set up in the tissues surrounding a tumor which may precipitate sudden paralysis.

Although the onset of a monoplegia due to cortical, subcortical, or capsular causes is sudden, the reactions of degeneration do not come on for a long period of time in such cases, because the muscles in the paralyzed area are still connected with the trophic centres in the cord, and this affords us a valuable point in differential diagnosis.

Sometimes a suddenly developed monoplegia affecting the arm comes on as a manifestation of hysteria, and follows the type of true cerebral hemorrhage so closely as almost to defy diagnosis. This condition may be accompanied by hysterical œdema, the hand becoming puffy and swollen. The presence of a neurotic temperament and other hysterical signs, coupled with the prompt development of contractures and the fact that the muscles do not rapidly waste, point to the cause of the loss of power in some cases, and this is emphasized if the presence of hysterical anæsthesia of the skin can be discovered. Further, if the hand is affected, Patrick asserts that in making an attempt to grasp an object the thumb and forefinger are chiefly used; but if the object is placed suddenly in the

ulnar part of the hand, the remaining fingers can grasp it easily. (See chapter on the Skin.)

In all cases of brachial monoplegia due to peripheral lesions we find that atrophy of the muscles comes on very rapidly from cutting off of the muscles from their trophic centres in the spinal cord.

Brachial monoplegia is nearly always the result of injury to the brachial plexus or to some of its important branches. The symptoms consist in heaviness or numbness of the arm with more or less loss of power. The motions of the arm which are particularly affected are usually abduction and elevation, which movements depend upon the circumflex nerve. If the power of extending the arm is lost, the loss depends upon paralysis of the musculo-spiral, which supplies the triceps; whereas if the power to flex the forearm is lost, there is paralysis of the musculo-cutaneous, which is the supply of the brachialis anticus and biceps. If the supinator longus is involved, the musculo-spiral is also affected.

When brachial monoplegia depends for its existence upon primary brachial neuritis there is pain in the wrist and hand at first, or on the scapula and in the axilla, thence radiating down the arm. This pain is constant and dull, and now and then excruciating, and is made worse by movement, even when the loss of power is comparatively slight. Sometimes, on the other hand, when the neuritis is septic in origin, it may start in the ulnar nerve and gradually extend up to the plexus. In still other cases brachial monoplegia may depend upon fracture or dislocation of the head of the humerus, and in such a case the paralytic symptoms are apt to be very well developed. The musculo-spiral nerve is often paralyzed by fracture of the humerus, and this results in paralysis of the muscles of the back of the arm and forearm and back of the hand, and of the skin covering these parts. Sometimes in locomotor ataxia the peripheral nerves seem quite as much involved as the spinal cord, and symptoms precisely like the paralysis of a toxic neuritis develop. Thus, Strümpell has reported cases of musculo-spiral paralysis from this cause, and Remak and Hirt record cases in which the median nerve has been affected, so that not only loss of power but wasting of the muscles has resulted. This is particularly the case if the muscles are much used in the daily pursuits. The ulnar nerve may also be affected. Such cases are separated from pseudo-tabes by the pupillary reflexes and other pathognomonic ataxic symptoms. (See chapter on the Feet and Legs and that on the Eye.) There are

still two comparatively rare forms of brachial monoplegia of the plexus type, namely, that due to pressure of growths in the neck, or axilla, and brachial paralysis of the upper arm type, sometimes called Erb's paralysis. This latter form occurs from paralysis of the fifth and sixth cervical nerves or their roots. In adults this commonly results from blows or heavy weights striking on the shoulder, and in infants from pulling on the neck in difficult labor. As already said, it is an upper arm palsy and is due to the loss of nerve-supply to the deltoid, biceps, brachialis anticus, and supinator longus and brevis, and the supra- and infra-spinatus muscles. The adult form is often associated with anæsthesia and is persistent. In infants it is often temporary and sensory symptoms are commonly absent.

When the lower arm is paralyzed as the result of trouble in the brachial plexus the lesion is in the nerves arising from the seventh and eighth cervical and first dorsal roots, and the muscles affected are the triceps, the flexors of the wrist and fingers, the pronators of the hand, the extensors of the fingers, and the muscles of the hand. The arm can still be raised by the deltoid and the forearm flexed on the arm.

When there is wasting with paralysis of the thenar, hypothenar, and interossei muscles, not due to progressive muscular atrophy, with anæsthesia in the arm and forearm in the part supplied by the ulnar nerve, and in addition myosis on the side of the lesion, with sluggish pupil, retraction of the eyeball, and partial closure of the lids, there is probably a lesion of the first dorsal root of the brachial plexus and the communicating branch of the second dorsal. The cause may be neuritis or pressure by a tumor. This form is sometimes called "Klumpke's paralysis."

Widespread muscular atrophy of the arm sometimes takes place in locomotor ataxia as a result of a coincident neuritis.

The presence of bilateral brachial monoplegia should always make the physician suspicious of lead-poisoning or crutch-paralysis.

(For a description of the areas involved in the spinal cord, which cause loss of power in the arms and legs, see chapter on Feet and Legs, part on Paraplegia, and tables of localization in that chapter, also plates in chapter on Skin.)

Apparent brachial monoplegia, in reality a syphilitic pseudo-palsy, has been described particularly by Parrot. A child apparently perfectly well, and but a few weeks old, suddenly loses the power of its arm, so that the member hangs like a flail. No wasting takes place,

no degenerative reactions occur, there may be some pain, and crepitation on moving the arm. The cause of these symptoms lies in the fact that there has been a separation of the epiphyses from the shafts of the bones with consequent helplessness. Sometimes general paralysis of the extremities arises from the extension of the disease to other limbs. The prognosis as to life is bad.

It yet remains for us to discuss the paralysis of several important groups of the muscles of the arm. If the forearm cannot be flexed, there is loss of power in the biceps and brachialis anticus, and to some extent in the supinator longus; and as the first two muscles are supplied by the musculo-cutaneous and the third by the musculo-spiral, such a failure in flexion shows paralysis of these fibres.

Paralysis of the extensors of the forearm, wrist, and hand, and of extension of the elbow with wrist-drop in consequence, and flexion of the tips of the fingers is due to disease affecting the musculo-spiral nerve, but the fingers can still be partly extended through the action of the interossei and lumbricales, provided the tips are flexed. The back of the hand and wrist become unduly prominent after a short time because of the forced flexion of the hand and rapid wasting of the extensors. In most cases the supinator longus, which supinates the forearm after it is pronated, is paralyzed. When the ability to pronate the forearm is greatly impaired, and the thumb is extended and abducted, so that it cannot be brought in contact with the tips of the fingers, the trouble is probably paralysis of the median nerve, and this is confirmed if all the phalanges are paralyzed except the first.

If the arm cannot be moved outward, away from the body, there is paralysis of the deltoid supplied by the circumflex nerve. In this connection attention should be called to the loss of power with wasting of the muscles seen after direct blows on the muscle or after injuries to the joint, sometimes called "joint-palsies."

Brachial Paræsthesia. Disturbances of sensation in the hand and arm consist in anæsthesia, analgesia and numbness, tingling, and pain. The area of these sensations depends upon the nerve-trunks involved, and to some extent upon the degree of involvement. Thus, if the function of the nerve is merely impaired, the sensation may be that of tingling or pain; if still further impaired, the sensation may be that of numbness; and if the sensory fibres be totally destroyed or paralyzed, absolute anæsthesia and analgesia may be present (see chapter on Skin Anæsthesia).

CHAPTER III.

THE FEET AND LEGS.

The general appearance of the feet and legs when clothed—The gait—Spastic paraplegia—Paraplegia without spastic contraction—Crural monoplegia—Deformities of the feet—The joints—Alterations in the nutrition of the feet and legs aside from a change in the muscles.

As the physician sees a patient approaching him, he can often gain information as to the ailment from which the man is suffering by noticing his gait and the appearance of the legs and feet, for, while the gait varies greatly in normal individuals, in some diseases it is so typical that he who runs may read the diagnosis. A glance at the feet revealing one foot more loosely covered than the other, or a slit in the shoe, or a very loose lacing, will point to the presence of some inflammatory or dropsical swelling, which forces the patient to give it room; and if the legs of a man of ordinary build look swollen and fill the trowsers tightly, while a glance at his face reveals that it is puffy, rather than one which is obese, dropsy, still more widespread, is probably the cause.

Gait. Aside from local injuries causing a lame gait, which will be found discussed in a book on surgical diagnosis by the writer's friend, Dr. Martin, we find that gout, rheumatism, and sciatica are the common causes of a limping gait, arising from trouble in one leg, and that in such cases there is a pained expression of the face at each movement, which shows the suffering that walking causes. The gait of such a patient is slow and cautious, and he is apt to rest every few steps, bearing his weight at such times chiefly on the well leg or, by means of his hands, upon chairs or tables that may be near. Aside from the alterations of gait produced by these causes, we see very typical gaits produced by locomotor ataxia, pseudo-locomotor ataxia (peripheral neuritis) due to alcoholic or lead-poisoning, syphilis, or peripheral neuritis arising from other causes, Friedreich's ataxia, general paresis, chronic myelitis, lateral sclerosis, acute poliomyelitis, pseudo-muscular hypertrophy, cerebral infantile palsy, multiple sclerosis, paralysis agitans, cerebellar disease,

organic and hysterical hemiplegia, and osteomalacia, and the gaits caused by rickets or other bony defects.

In locomotor ataxia the gait is unsteady and waveringly uncertain, resembling that of a blindfolded person who is told that he is approaching some inequality in the floor. The patient continually seems to be feeling for the ground with his feet, and carefully picks his way along a perfectly smooth surface in a labored fashion, using a cane to help him both in the way of support and of feeling the ground. If he looks up from the ground while walking, he sways suddenly and may fall, and if prevented from returning his eyes to the pavement almost surely falls if no aid is given him. (Fig. 26.)

Fig. 26.

Gait in a case of locomotor ataxia. From instantaneous serial photographs of a patient of Dr. Dercum, made simultaneously from two different points of view by Muybridge.

The gait of pseudo-tabes is sometimes identical with that just described, is usually associated with a history of alcoholic excess, and is due to multiple neuritis. In a majority of the cases, however, it is distinctive, and has been called the "steppage" gait. The foot is thrown forward and the toe is raised so that the heel first strikes the ground in much the manner adopted when one attempts to step over some obstacle. Sometimes this gait is found in cases of arsenical neuritis and that due to lead, but in alcoholic tabes there are generally

mental symptoms associated with this gait, while in lead-poisoning the pathognomonic signs of this condition, such as the blue line on the gums and wrist-drop, when combined with the history, clear up the diagnosis. It must not be forgotten, however, that the differential diagnosis of tabes from pseudo-tabes is sometimes very difficult, and as Dana has well said : "When Déjèrine described as locomotor ataxia a case which now appears to have been one of alcoholic peripheral neurotabes, when Buzzard has diagnosticated as true spinal tabes a case of post-diphtheritic ataxia, when Seligmueller mistakes a case of wall-paper-poisoning for one of true spinal tabes, we may easily suppose that errors have been made by many others."

The important symptoms which point to true locomotor ataxia are the swaying of the body when the eyes are closed (Romberg's symptom), the loss of knee-jerk (Westphal's sign), the history of gastric, laryngeal, or vesical crises, the presence of numbness in the feet, the slow onset of the disease, and the absence of any history of exposure to the causes of neuritis just named. If all these signs are present, and are combined with that most important symptom, the Argyll-Robertson pupil, the diagnosis is practically certain.

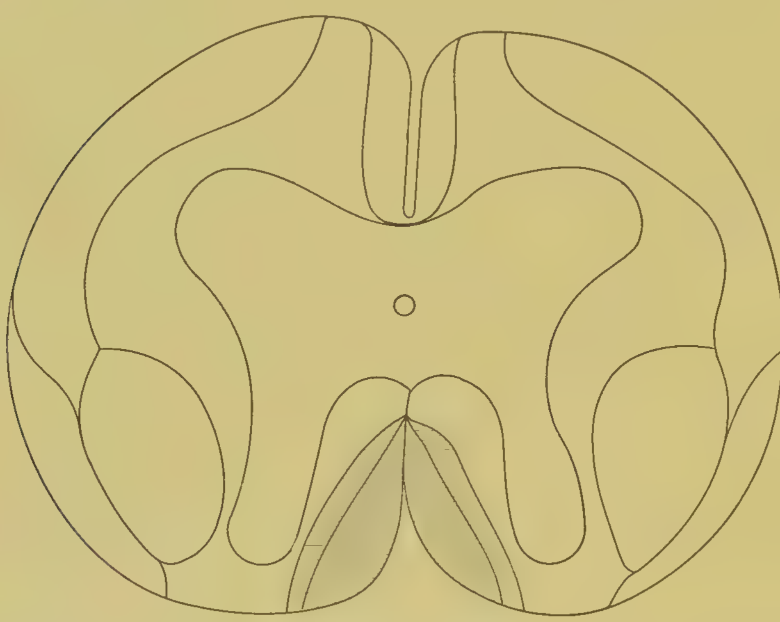
The following table from Peterson's article in Dercum's *Nervous Diseases* shows very clearly and comparatively the symptoms of the first, second, and third stages of true locomotor ataxia.

Initial Period.	Second Stage.	Final Stage.
Inco-ordination, but no change of gait.	Greater inco-ordination, and marked ataxic gait.	Cannot walk because of ataxia.
Numbness of the feet.	More marked anæsthesias.	Extensive anæsthesia.
Shooting-pains in the legs.	Pains worse.	Pains less.
Diminished or lost knee-jerks, one or both.	Lost knee jerks.	Lost knee-jerks.
Sluggish or lost pupillary reflex to light.	Lost pupillary reflex to light and myosis.	Lost reflex to light, myosis, paralysis of accommodation.
Weakness of sexual function.	Impotence.	Impotence.
Transient diplopia ; transient ptosis.	Ocular palsies rare, or marked ophthalmoplegia.	Ophthalmoplegia.
Sluggish micturition.	Increased vesical weakness.	Catheterization needed.
Optic atrophy.	Optic atrophy rarely develops.	Blindness.
Trophic changes in the joints.	Trophic changes not so common.	More marked if they began in early stage.
Hemiatrophy of tongue.	Deafness.	Increased.
	Laryngeal and visceral crises.	Not so common.
	Girdle sensation.	Unnoticed.

In neuritis causing pseudo-tabes we have a history of rapid onset of the symptoms, paralysis and wasting of the muscles, and *an absence of vesical symptoms and the Argyll-Robertson pupils.*

Reflex action is decreased and the gait altered in locomotor ataxia, because, though the motor tracts are open, the sensory tracts in the nerves, the posterior nerve-roots, and the posterior columns of the cord are diseased. For these reasons the reflex arc is destroyed and the co-ordination of the muscles lost. The patient cannot tell how to use his muscles unless he can see them and co-ordinate them by the aid of the eye. The sensations of formication or numbness are also due to these sensory lesions. (Fig. 27.) (For description of motor and sensory tracts of the spinal cord, see early part of chapter on Hemiplegia, and chapter on the Skin.)

Fig. 27.



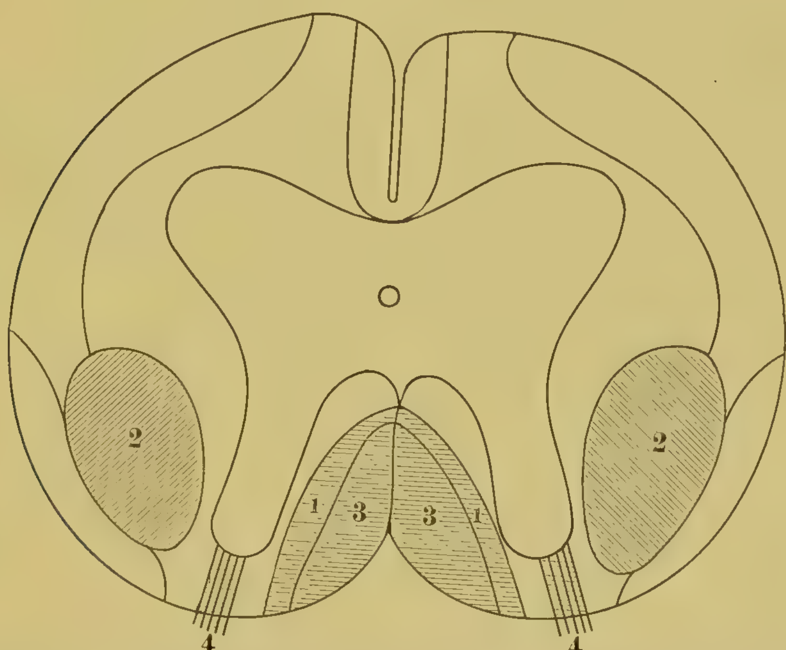
Cut showing the areas of the cord involved in locomotor ataxia. The shading includes both the column of Goll, the inner, and that of Burdach, the outer. It is to be remembered that the lesions of locomotor ataxia are found in the peripheral nerves as well.

Sometimes not only the gait, but the entire set of the ordinary symptoms of locomotor ataxia are aped by hysteria so closely that a diagnosis may be almost impossible, but the Argyll-Robertson pupil, the lost knee-jerks, and the optic atrophy will not be present if hysteria be the cause of the symptoms. On the other hand, Romberg's symptom may be marked to an extraordinary degree. The patient who is hysterical, in falling nearly always falls the same

way, keeping her frame stiff like a board. (See chapter on Eye for differential ocular symptoms.)

In Friedreich's ataxia the gait is peculiar. The legs are widely separated and moved in an uncertain, hesitating manner, and if the feet are placed close together and the patient told to stand still swaying at once develops. If the eyes be closed, the swaying may greatly increase, and the movements of the arms are inco-ordinated. These symptoms, which to a certain extent simulate true locomotor ataxia, are associated, as a rule, with others which separate the two affections, for in this disease the symptoms often come on in very early life, there is sometimes nystagmus, usually a history of heredity, there is a slow and jerky articulation, scoliosis, talipes equino-varus, but there is no Argyll-Robertson pupil. (Fig. 28.)

FIG. 28.



Cut showing the spinal areas chiefly involved in Friedreich's ataxia. The areas are the column of Burdach (1); the lateral pyramidal tracts (2); the columns of Goll (3); the posterior nerve-roots (4).

Friedreich's ataxia must be separated from another rare disease in which the gait is ataxic and the disease hereditary, namely, hereditary cerebellar ataxia, in which we have the following symptoms not seen in Friedreich's disease, namely, normal or exaggerated knee-jerks, Argyll-Robertson pupils, and a beginning of the malady after twenty years of age. The following table compiled by Collins, of New York, gives the differential points between these diseases:

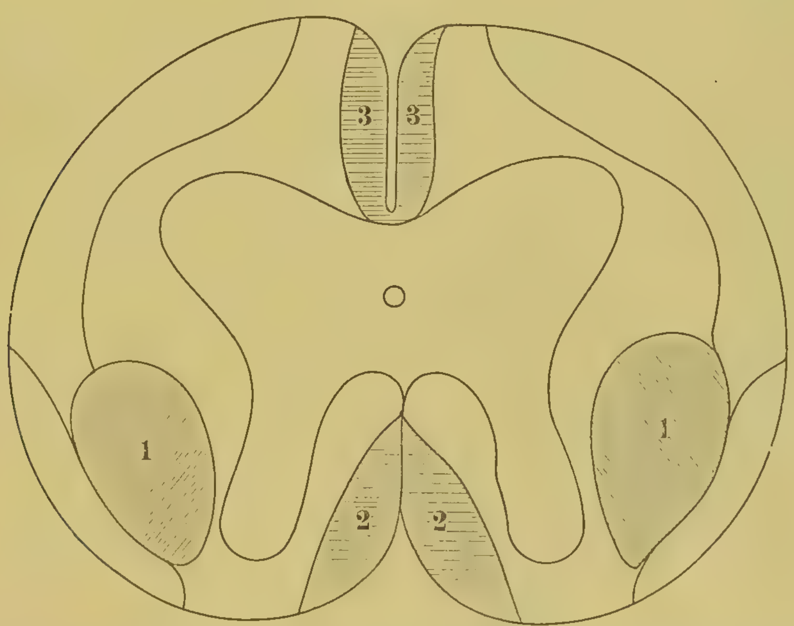
Hereditary Spinal Ataxia. *Friedreich's disease.*	Hereditary Cerebellar Ataxia.
1. Gradual impairment of co-ordination, first in legs, afterward in arms. Later in the disease the patient may reel as if under the influence of alcohol. A quick backward and forward balancing movement.	1. Gait: Uncertain, reeling; gait of one inebriated. Patient frequently walks with body bent forward and head thrown backward, and the feet wide apart. Does not have to watch the feet.
2. Station: Closure of eyes, as a rule, increases the unsteadiness; this may be absent.	2. Station: Romberg symptoms absent.
3. Titubation of upper extremities very uncommon. Irregularity in voluntary movements of arms and fingers.	3. Titubation and inco-ordination and loss of dexterity in the upper extremities. Choreiform movements exaggerated on voluntary effort; "intentional."
4. Frequently jerky, irregular movements of head and neck. Sometimes like an irregular tremor.	4. Not infrequently oscillations or jerky movements of the head, less often of the trunk.
5. Mimetic muscles do not show ordinarily overcontraction.	5. Exaggerated contraction of the mimetic muscles on speaking.
6. Ataxia is not so great when the patient is lying.	6 Ataxia is very much less, or disappears when the patient is lying down, but the inco-ordination persists.
7. Affection of speech may be absent; when it does occur is a late symptom, and consists of an eliding of syllables and an occasional hesitation.	7. Speech: Hesitating, abrupt, explosive, ataxic, defective.
8. Nystagmus is a very common symptom, but it may be lacking.	8. Eyes: Twitching of the eyeballs very common, but not nystagmus. Optic atrophy, progressive choroiditis, paralysis or paresis of the external recti sometimes.
9. Myotatic irritability is lost. Knee-jerks may be present in the beginning of the disease, but they soon disappear. Ankle-clonus is never present.	9. Myotatic irritability increased; reflexes exaggerated, such as knee-jerks; often ankle-clonus.
10. Mentally, normal. Very rarely any defect.	10. Mental shortcomings varying from slight psychical disturbances up to a considerable degree of dementia.
11. Deformities of the extremities such as *pied bot* and spinal curvature, very common.	11. Deformities of the extremities and spine, such as *pied bot* or scoliosis, do not occur or are most rare.

The feebleness of the limbs, the reflex iridoplegia (Argyll-Robertson pupil), and the ataxic gait sometimes seen as the chief manifestations of *general paresis* may cause an error in diagnosis in favor of locomotor ataxia, but careful examination will reveal mental feebleness in the paretic case or at least evidences of delusions, and if the disease is at all advanced there will be a history of the patient having had convulsions or apoplectiform attacks. Sometimes there will be found present in paretic dementia increased knee-jerks and many of the symptoms of ataxic paraplegia, but the associated mental failure and fine intention-tremor of the hands decide the diagnosis in favor of paretic dementia.

In chronic myelitis in the early stages, while motion is still preserved the gait is typically that of feebleness, and the legs respond slowly to the cerebral desires, being dragged along after the patient,

who leans forward, supporting some of his weight on crutches or canes. If the lesions of the disease involve the lateral pyramidal tracts to a considerable extent, the gait is somewhat spastic, while if the sensory fibres are much involved it may be like that of ataxia. Under these circumstances the attitude and gait of a patient are sometimes a combination of those of lateral spinal sclerosis (spastic paraplegia) and locomotor ataxia. In some instances the spastic symptoms are more marked, in others the signs of locomotor ataxia are more prominent. This condition is called ataxic paraplegia, and in it we find the exaggerated knee-jerks of lateral sclerosis associated with the swaying of the body (Romberg's symptom) of ataxia. Ankle-clonus is also present. The crises of locomotor ataxia do not occur, and the Argyll-Robertson pupil is usually not present. (Fig. 29.)

FIG. 29.



Cut showing areas of spinal cord involved in ataxic paraplegia, which is practically a combination of locomotor ataxia and lateral sclerosis. 1. Lateral or crossed pyramidal tracts. 2. Posterior columns of Goll and Burdach. 3. Direct pyramidal tracts or Türck's columns.

In lateral sclerosis the gait is typically spastic, the legs being rigid from the hip-joint down, and the toe being dragged in a semicircle from behind forward.

When the gait of a young child is stumbling, or the leg or legs are dragged after it, or the ankles bend so that locomotion is impossible, the probable diagnosis is that the cause is acute poliomyelitis (see Paralysis of Leg).

In pseudo-muscular hypertrophy there is a peculiar waddling gait,

a tendency to stumble, the body is usually bent backward, and there is difficulty in getting up from the floor and on going up and down stairs. The patient in all his movements shows a marked loss of power in the legs with a great apparent increase in the size of the muscles in the legs. (Fig. 30.)

FIG. 30.

Typical pseudo-muscular hypertrophy. (DERCUM.)

The gait of pseudo-muscular hypertrophy is sometimes closely reproduced in children suffering from severe rickets, and the other features of the case which may mislead the physician are that the child, if fat, will have bulging legs, as if the muscles were hypertrophied, and lordosis due to spinal weakness. In the rickety case, however, the knee-jerk is preserved, and in the pseudo-muscular hypertrophy it is lost.

The gait of a child suffering from infantile cerebral paralysis is quite characteristic. In the first place, it is spastic, and the patient walks on the toes or in some cases club-foot develops. The heels are everted and the toes turned inward, the knees being so closely approximated that the clothes may become worn between them from the rubbing. So great is the extension of the legs that the toes are very apt to drag, and, finally, the adduction spasm may be so great that the legs overlap each other as walking is attempted. (Fig. 31.)

The gait in multiple sclerosis is often markedly spastic and paretic —that is, stiff and feeble, and may in the early stages of the disease closely resemble that of spastic paraplegia due to lateral sclerosis. When the patient attempts to pick up a small object with his fingers

first noticed when the child attempts to walk. Cerebral spastic paraplegia in infants also sometimes comes on in cases of so-called arrested development. Such cases present no abnormality for the first few months of life, then cease to develop in mental brightness, fail to recognize the nurse or mother, cease to play, gradually lose their vision, and develop nystagmus. Death usually takes place in one or two years at the latest. Convulsions do not occur in this state, but tremors are often present in the arms. There is no history in such cases of difficult labor or premature delivery. In both this and the infantile cerebral form of spastic paraplegia the pyramidal tracts are degenerated. It is important to remember that cerebral paraplegia is not associated with the development of the reactions of degeneration in the paralyzed part, and is associated with comparatively little wasting, thereby differing from the deformities of the lower extremities resulting from poliomyelitis or acute infantile palsy.

Care should be taken that the spastic paraplegia of rickets is not mistaken for a birth-palsy.

A cerebro-spinal cause of spastic paraplegia in adults is multiple cerebro-spinal sclerosis, in which condition the loss of power amounts to a paresis rather than an absolute paralysis. The presence of intention-tremors, exaggerated knee-jerks and ankle-clonus, nystagmus, and vertiginous, epileptiform, or apoplectiform seizures, with staccato speech, and local areas of loss of power elsewhere, associated with spastic paraplegia, renders the diagnosis easy.

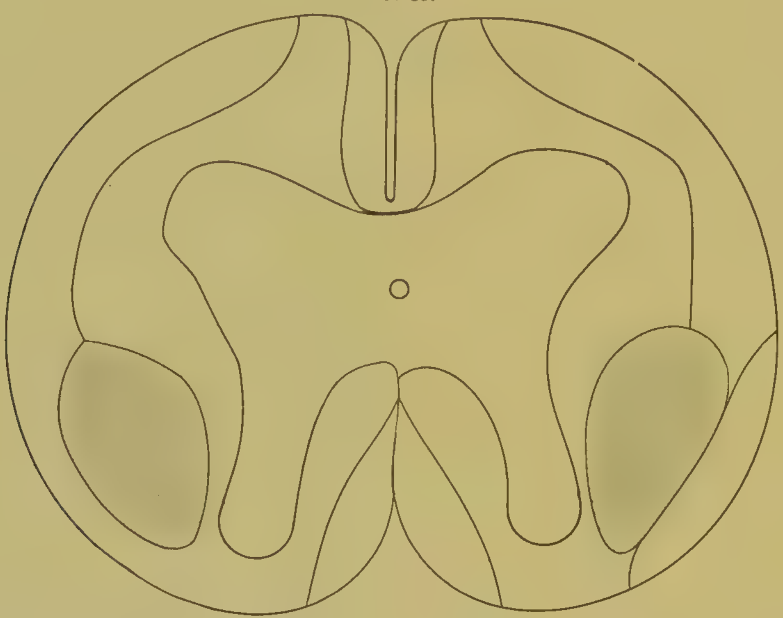
The *spinal* lesions giving rise to paraplegia of the lower extremities are numerous, and are perhaps best grouped in the following table of Bramwell:

1. Organic disease . . .	Inflammation of cord	Medullary.
	Softening " "	
	Hemorrhage " "	
	Tumors " "	
	Meningitis " "	Meningeal.
	Meningeal hemorrhage	
	Injuries	
	Tumors	
	Caries of bone	Osseous.
	Tumors of bone	
2. Functional	Hysterical.	
	Reflex.	
	Malarial and anæmic.	
	Dependent on idea.	

The natural sequence is to pass on to a consideration of the forms of spastic spinal paraplegia, and to take up first of all its manifestation in children. This occurs in what is known as hereditary spastic

paralysis, which is to be separated from infantile cerebral paralysis by the absence of a history of injury to the head at birth, and the absence of convulsions and defective mental development, all of which appear in the cerebral form, and this absence of convulsions and defective mental power in this form of spastic paraplegia almost certainly separates it from the cerebral infantile type of paralysis. It is to be separated from the spastic paraplegia of lateral sclerosis by the fact that it occurs in early life, and that there is a history of heredity, or of several members of the family being affected by the disease. There are usually rigidities and contractures, but the bladder and rectum escape the paralysis, and there are no trophic changes. The reflexes are increased. This disease is rare.

FIG. 35.



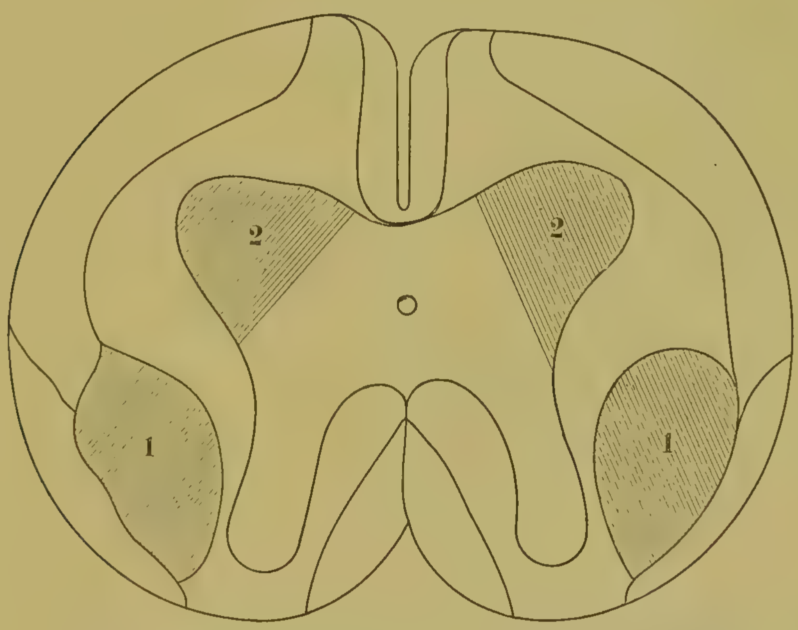
Cut showing shaded areas involved in lateral sclerosis, viz., the crossed pyramidal tracts.

In transverse myelitis there is often in the later stages of the malady spastic paraplegia as a result of the irritability of the spinal centres below the seat of the lesion, and this may cause a spastic gait. In distinction from lateral sclerosis, we find in myelitis that there are a girdle pain, involvement of the bladder and rectum, and sensory paralysis.

In the adult, when there is loss of power in the lower limbs with spastic contraction of the muscles when the patient attempts to move them, so that they become rigid, or if before the stage of rigidity develops the gait is spastic and stiff and the reflexes are greatly exaggerated, the disease is generally lateral spinal sclerosis. (Fig. 35.)

There is also in lateral spinal sclerosis absence of both sensory disorders and rectal and bladder troubles, but sometimes there is present excessively hasty urination. The reason why the reflexes are increased in lateral sclerosis, and similar ailments associated with spastic paraplegia, is that the inhibitory fibres which descend from Setschenow's reflex inhibitory centre in the medulla oblongata are destroyed in the lateral pyramidal tracts. In amyotrophic lateral sclerosis similar symptoms associated with wasting of the muscles are present in the later stages, but in the early stages the arms are chiefly affected by the wasting and paralysis. (Fig. 36.) (See chapter on Hand and Arm.)

FIG. 36.



Cut showing area of spinal cord involved in amyotrophic lateral sclerosis. 1. Crossed pyramidal tracts. 2. Anterior horns of gray matter containing the trophic cells.

Spastic paraplegia may also be due to spinal pachymeningitis, and the associated symptoms may so closely resemble those of myelitis that a diagnosis is impossible, but the spastic character of the paraplegia, the early appearance and severity of the pain, and the comparatively slow development of the symptoms in pachymeningitis, will aid in separating the two affections, as will also the presence of persistently increased reflexes from the first. Sensory disturbances, aside from pain, are common in myelitis, but rare in this condition. If the inflammatory process becomes widespread, there may be sensory disorders and trophic sloughs owing to invasion of the por-

tions of the cord connected with sensation and nutrition by a secondary myelitis. The development of signs of spinal caries in such cases at once shows the condition to be meningeal in origin, and the history of traumatism will point to meningitis rather than myelitis.

Spastic paraplegia, greatly increased tendon-reflexes, low muscle-tension, vesical disorder, and slight sensory disturbances should make the physician think of spinal syphilis.

NON-SPASTIC PARAPLEGIA. Passing from spastic paraplegia we come to those forms of paraplegia lacking this peculiarity. They are quite numerous and important. If the paraplegia comes on suddenly, the cause may be hemorrhage into the substance of the cord or into the spinal membranes, or be due to compression or destruction of the cord by injuries to the back, whereby there is laceration of the soft parts or fracture or dislocation of the vertebræ. When the paraplegia is slower in onset the spinal causes are acute ascending paralysis or Landry's paralysis, acute central myelitis, and acute transverse myelitis. On the other hand, the slowly oncoming paraplegias are due to chronic myelitis, to locomotor ataxia, amyotrophic lateral sclerosis, lateral sclerosis, poliomyelitis, neuritis, and pressure due to disease of the vertebræ or to spinal tumors. Finally, we have what are called reflex and hysterical paraplegias.

Hemorrhage into the spinal cord is an exceedingly rare condition unless preceded by grave disease of its tissues. Indeed, the existence of such a condition in man has been denied. The patient previously in good health is stricken suddenly to the ground, and there may be almost as much cerebral disturbance as in cerebral apoplexy, but consciousness is generally preserved. The total amount of paraplegia may be instant, or not be complete for twenty-four hours. Bedsores speedily develop, and death ensues from exhaustion or from extension of the hemorrhage upward to the vital centres. Practically identical symptoms ensue when the hemorrhage takes place between the membranes covering the cord. In both instances the reflexes are lost if the hemorrhage be sufficient to produce total paralysis.

If, on the other hand, after a prodromal period of short duration, during which there is some fever, the patient is suddenly attacked with paraplegia, the cause may be the acute ascending myelitis of Landry, and the rapid extension to the trunk, the arms, and the respiratory muscles, with the consequent early death of the patient, will confirm the diagnosis. There is usually no involvement of sensa-

tion or trophic paralysis, and the sphincters of the bladder and rectum escape the paralysis. Similar symptoms associated with sensory disturbances are probably due to a polyneuritis.

Diller and Meyer state that the cardinal points for the differential diagnosis should be:

1. Flaccid paralysis of the muscles, spreading rapidly from one point over the rest of the body, generally beginning in the legs, but sometimes following the reverse order, as in the French zoölogist Cuvier.
2. Absence of muscular atrophy and of electrical reaction of degeneration.
3. Tendon and superficial reflexes absent.
4. Sensibility not, or only slightly, impaired.
5. Sphincters, as a rule, intact (exceptions rather frequent).

By far the most common cause of paraplegia is myelitis in one of its forms, but, whether the onset be rapid or slow, it must be remembered that the symptoms of myelitis depend, first, upon the level at which the spinal cord is involved, and, second, as to whether the lesion involves the white matter or the gray. If the lesion is an acute central myelitis of the gray matter, it usually produces many of the symptoms about to be detailed under acute transverse myelitis, but the onset is malignant and the areas involved are usually widespread. It is attended by fever of a marked type, though the temperature of the paralyzed parts is below normal, and by early evidences of trophic lesions. Multiple arthritis may come on. The bladder and rectum are paralyzed, and, finally, delirium may develop. The prognosis is unfavorable. Acute central myelitis is to be separated from Landry's paralysis by the facts that in it sensation is lost, there are rectal and vesical paralysis, fever, and rapid trophic changes. From polyneuritis it is separated by the facts that there are no great trophic changes in this form of neuritis, and the rectum and bladder are rarely paralyzed.

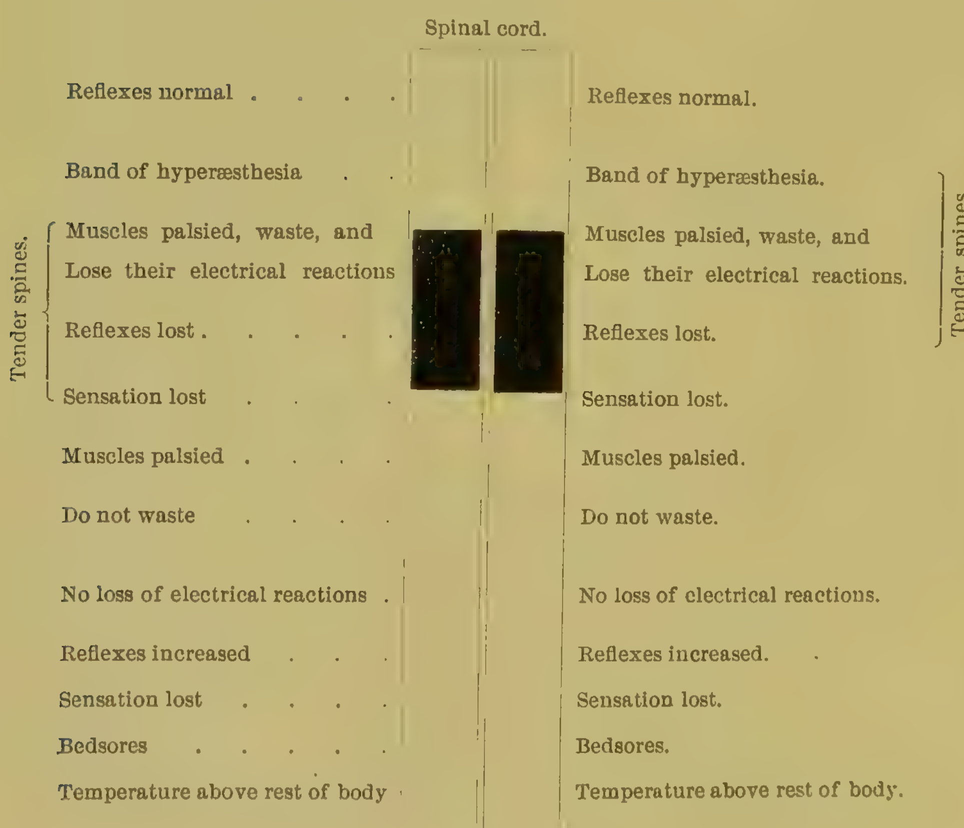
The symptoms of acute transverse myelitis are capable of being divided into three groups, in the first of which the onset is as sudden as is that of an apoplexy, in the second the symptoms come on quickly, and in the third more subacutely. In the acute forms, however, the history will be that after a period of numbness, heaviness, and weakness of the legs, with more or less pain in the back, the patient has found it impossible to move his legs, has lost control of his bladder and rectum, or suffers from retention of the urine and

feces instead, and at the same time has developed anæsthesia of his lower extremities, and the girdle sensation, or, if the lesion be situated high up in the cord, tingling in his arms (see chapter on Skin). The reflexes may be abolished at first, and then return in an exaggerated form in the segments of the cord below the area affected. In other cases the reflexes do not return if the lesion is completely transverse. The patient is speedily bed-ridden, and to these symptoms just detailed are soon added the development of bedsores and sloughs on dependent parts of the legs or on the buttocks, followed, it may be, by death from exhaustion, although the case may survive for months and even become somewhat better. If improvement takes place, sensation returns in the course of from one to six months, some motion in from six to eighteen months, and, finally, spasms and contractures may result from descending degeneration of the lateral tracts.

The following diagram from Taylor's *Index of Medicine* shows the effect of a lesion in the spinal cord in transverse myelitis.

Symptoms in Transverse Myelitis.

The darkened portion represents the seat of lesion.



In cases in which paraplegia results from the more subacute form of transverse myelitis the symptoms are usually not quite so rapid in their onset as in the type just named. The patient first notices that his bladder and rectum are unduly irritable, and in his limbs there may be subjective sensory disturbances (see Paræsthesia in chapter on the Skin). The motor symptoms begin by a feeling of heaviness or inability to move quickly the lower limbs, so that the patient feels tired on slight exertion. Soon these symptoms deepen into absolute anæsthesia and motor paralysis, and the girdle sensation on the trunk becomes well developed (see chapter on Skin). The bladder, which at first was irritable, may now be toneless, paralyzed, and retentive or incontinent. Retentive, if the lesion is above the lumbar cord; incontinent, when the lower part of the lumbar enlargement is diseased. The reflexes may at first be abolished, but very soon some of them return, only those reflexes the centres for which are destroyed by the transverse lesion being abolished; that is, the reflexes recover after the first shock of the attack, and those muscles and tendons having spinal centres below the lesion have their reflexes increased because they are cut off from the inhibiting centres higher up in the cord or medulla. The muscles of the legs, which at the first shock of the onset of the malady were all flaccid and paralyzed, now divide themselves into two classes, those that are connected with the diseased part of the cord, which remains paralyzed, and those which are connected with the lower centres, which recover some power; but as the lesion is so placed as to cut them all off from cerebral influences, voluntary motion is lost as completely as if all were deprived of spinal influence. The truly paralyzed muscles waste, but the others which have unimpaired spinal centres do not, except very slowly from disuse. On the contrary, they often become spastically contracted. Other trophic changes, such as bedsores and bullæ, develop in the skin connected with the diseased focus, but not in that connected with centres below the lesion. Anæsthesia is present because the lesion prevents the sensory impulse from reaching the brain (see chapter on Skin). When the entire cord is not evenly involved in the transverse lesion certain groups of muscles partly escape. It is asserted that the extensors escape oftener than the flexors. The height of the paralysis also depends upon the situation of the lesion in the cord, and if high enough to involve the cervical region, and yet not high enough to paralyze the diaphragm and cause death (third or fourth cervical), there may be contraction

of the pupil by involvement of the fibres from the nucleus of the third nerve, which runs down the cord to the last cervical vertebræ before joining the sympathetic. When the legs become spastic late in transverse myelitis the cause is supposed to be a descending degeneration in the pyramidal tracts.

The symptoms of chronic transverse myelitis producing paraplegia are practically identical with the more acute form just described, except that they are very slow in their development.

Having discussed the various forms of myelitis, we have still to study the question as to the seat of the lesion in each form. Let us suppose that a patient presents himself with the following condition : There is complete paralysis of his arms and legs, with paralysis of the muscles of the trunk, and total anæsthesia of the same areas. The legs are in a state of spastic paralysis, their reflexes are increased and their nutrition is unimpaired; while the arms are found relaxed and flaccid, devoid of reflex excitability, and undergoing degenerative atrophy. The bladder and rectum are not retentive. All these symptoms point to a transverse lesion of the spinal cord in the cervical region, probably between the fifth cervical and first dorsal vertebræ.

If, on the other hand, the upper extremities are not affected (except, perhaps, the small muscles of the hand), but there is the same loss of power in the legs, with spastic contraction of the muscles, and the other symptoms just named are present, combined with degeneration of the muscles of the trunk, the lesion is probably somewhere between the second and twelfth dorsal vertebræ.

Again, if the paralysis of motion and sensation be only in the lower limbs, and there be flaccidity of the muscles (where before we discovered spastic contraction), with muscular degeneration, loss of reflexes, and paralysis of the bladder and rectum, the lesion is in the lumbar cord.

Still further, if there be loss of power with degeneration of the small muscles of the feet, and loss of sensation of the outside of the feet and toes, and of the skin about the anus, with preservation of power in the thighs and of the patellar reflex, the lesion is in the sacral cord.

Finally, it is possible for disease of the cauda-equina to produce symptoms of a lumbar-sacral lesion owing to the fact that this part of the cord is composed of fibres derived from these two areas. The patellar reflex may be preserved as the lesion is below the reflex arc, and all the fibres may not be involved.

Differential Diagnosis of Lumbar, Dorsal, and Cervical Myelitis.[1]

	Lumbar myelitis.	Dorsal myelitis.	Cervical myelitis.
Paralysis.	Paraplegia.	1. Dorsal, abdominal, and intercostal muscles, according to height of lesion. 2. Legs.	Neck muscles, diaphragm, arms, trunk, and legs.
Sensation.	Pains in legs, or girdle pains around loins; hyperæsthetic zone around loins; anæsthesia of legs, complete or uneven distribution.	Girdle-pain and hyperæsthetic zone between ensiform cartilage and pubes.	Hyperæsthesia and pains in certain nerve-distributions of arms; below this anæsthesia of arms, body, and legs.
Atrophy.	Of legs.	Of dorsal and abdominal (and intercostal muscles not subject to examination) corresponding to height of lesion; sometimes mild and slow of legs.	Atrophy of neck muscles (rare) or more commonly of arms.
Electrical reaction.	R D. in atrophied muscles; or in mild cases quantitative diminution.	R. D. in dorsal and abdominal muscles; slight quantitative changes only in legs when wasted.	R. D. in atrophied muscles.
Bladder.	Incontinence from paralysis of sphincter.	Retention, or intermittent incontinence from reflex action; later from overflow. Cystitis common.	Same as in dorsal myelitis. .
Bowels.	Incontinence from paralysis of sphincter, disguised by constipation.	Involuntary evacuation from reflex spasm or constipation.	Same as in dorsal myelitis.
Reflexes superficial.	Lost	Temporary loss, then rapid increase.	Same as in dorsal myelitis.
Reflexes superficial.	Lost.	Temporary loss, then slow increase.	Same as in dorsal myelitis.
Priapism.	Absent.	Often present.	Often present.

In this connection the reader should compare that part of the chapter on the Skin which deals with anæsthesia.

This subject is still further subdivided and elucidated by the following table and by the illustration on page 99.

Localization of the Functions of the Segments of the Spinal Cord. (According to Starr.)

Segment.	Muscles.	Reflex.	Sensation.
II. and III. C.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium(?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV. C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra and infra spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of the neck.	Neck. Upper shoulder. Outer arm.

[1] From Prince's article in Dercum's "Nervous Diseases."

Segment.	Muscles.	Reflex.	Sensation.
V. C.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. 5th cervical to 1st dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm Outer side of arm and forearm, front and back.
VI. C.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII. C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII. C.	Flexors of wrist and fingers. Intrinsic muscles of hand.	....	Forearm and hand, inner half.
I. D.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.	....	Forearm, inner half. Ulnar distribution to hand.
II. to XII. D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric. 4th to 7th dorsal. Tickling mammary region causes retraction of the epigastrium. Abdominal. 7th to 11th dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerves. Upper gluteal region.
I. L.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 1st to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II. L.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patella tendon. Striking tendon causes extension of leg.	Outer side of thigh.
III. L.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.	....	Front and inner side of thigh.
IV. L.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. L.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.	....	Back of thigh, back of leg, and outer part of foot.
I. to II. S.	Flexors of ankle. Long flexor of toes. Peronei. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh. Leg and foot, outer side.
III. to V. S.	Perineal muscles.	Foot reflex. Achilles tendon. Overextension of foot causes rapid flexion; ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perineum. Genitals.

Paraplegia when due to locomotor ataxia is nearly always so surrounded by other typical symptoms of this disease as to render its separation from the paraplegia of myelitis easy, and, further, it is rarely a true loss of power. The stabbing and darting pains of ataxia (see chapter on Pain), the presence of the Argyll-Robertson pupil, the absence of the patellar reflex, and the atrophy of the optic nerve are all characteristic of ataxia, and absent in myelitis (see also early part of this chapter on Gait).

FIG. 37.

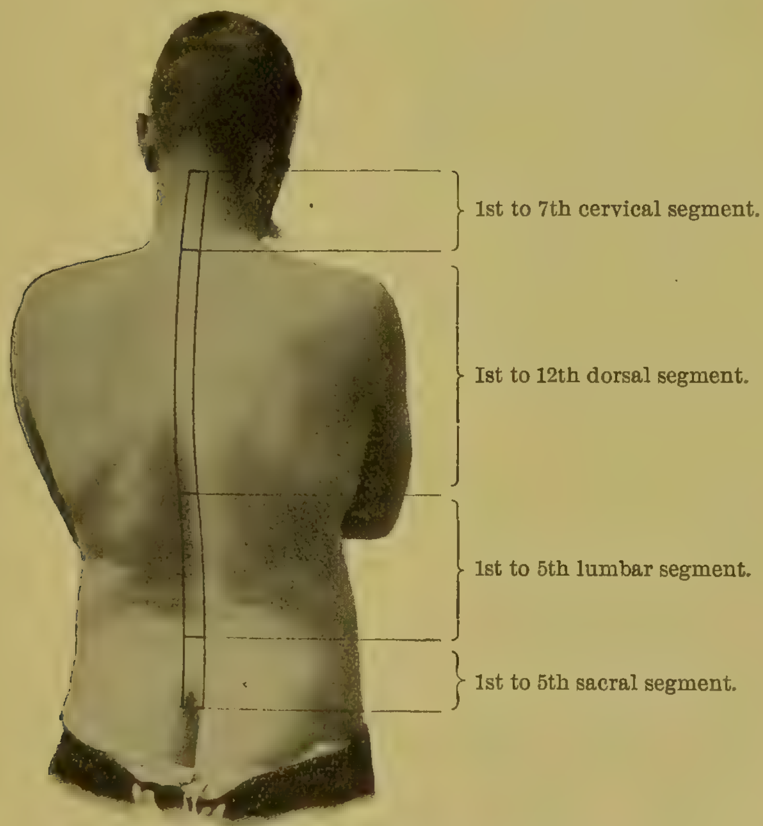


Diagram showing the surface-areas of the back corresponding approximately to the areas of the spinal cord supplying the trunk and limbs.

The symptoms of lateral sclerosis and amyotrophic lateral sclerosis have already been discussed under "Gait" and Spastic Paraplegia, but in the paraplegia called "ataxic paraplegia," also already discussed, there are in association lateral sclerosis and posterior sclerosis, and for this reason some of the symptoms of both are found to be present. Thus, in addition to loss of power there is a spastic condition of the legs with exaggerated reflexes, absence of the Argyll-Robertson pupil, and of crises of pain, but the Romberg symptom, or swaying

when the eyes are closed, is present. The condition which most closely resembles ataxic paraplegia is that of tumor of the middle lobe of the cerebellum, but in such cases we have, in addition, headache, vertigo, optic neuritis, titubation, and sometimes vomiting.

The onset of paraplegia in a young child, preceded by an attack of fever, vomiting, restlessness, and general illness, lasting but a few hours or days, and which may be complicated by convulsions, all point to the cause being poliomyelitis of a severe type. The legs are, however, as a rule, completely paralyzed for but a brief period after the attack. Eventually the storm clears off and only the muscles directly connected with the diseased cells in the cord (anterior cornua) remain paralyzed. There is no loss of sensation, but reflex action is abolished in the paralyzed parts. Far and away the most important point in the diagnosis is the symptom of rapid wasting of the muscles in the paralyzed parts and the rapid development of coldness in these areas, which is due to the destruction of the trophic centres in the spinal cord.

Paraplegia resulting from tumor of the cord or its membranes only ensues when the growth is so placed as to cut off all the motor tracts supplying both limbs, which is rarely accomplished until after a long history of more or less well-developed motor and sensory failure. The paralysis is developed in the areas supplied by the centres in the cord below or at the level of the growth, and the violent pain nearly always present in cases of tumor points to the diagnosis. Very painful paraplegia, therefore, indicates spinal tumor as its cause. The area of anæsthesia and the muscles involved may also give definite information as to the seat of the growth (see chapter on the Skin, and Starr's table just quoted.)

If the paraplegia be due to compression from fracture or dislocation of the vertebræ or to other direct injury, the history of the patient and the evidences of external local mischief will decide the diagnosis.

Sometimes during the course of severe disease, producing irritation of the bladder, kidney, bowels, or rectum, as in violent cystitis, stone in the kidney, and dysentery, paraplegia comes on, due in some cases to an infectious myelitis, but in others to what is apparently only a reflex paralysis, as it often passes away with the removal of the source of irritation. Even worms in the intestine have produced such a paralysis, and their removal has been followed by cure. Generally sensation in the limbs is unimpaired and the bladder and

rectum act normally. Sometimes, however, in the presence of severe renal disease, as renal calculus, there may be all sorts of disturbance of sensation and pain, as well as great motor paralysis, with total loss of reflexes, following an exaggeration of the reflexes. Probably these severe cases are always due to a coincident myelitis rather than to reflex irritative cause.

No form of paraplegia presents so many types or represents so many organic diseases as does that due to hysteria, for there may be not only great loss of motion but exaggerated or lost reflexes, relaxation or spastic contraction of the muscles, anæsthesia and hyperæsthesia, pain or no pain. The very occurrence of such irregular manifestations in a young neurotic girl, the fact that the anæsthetic areas constantly tend to shift their position, and, finally, that the contractures, if present from hysteria, disappear on administering an anæsthetic to a stage in which muscular relaxation is produced in the ordinary individual, aid us in making what is in some cases an almost impossible diagnosis (see that part of this chapter on Contractures).

A pseudo-paralysis of the legs with immobility sometimes occurs as a symptom of scorbutus in infancy. The parents notice that the child flinches when picked up or handled, and seems as if tender from rheumatism. Often the gums are swollen and bleeding, and purpuric eruptions appear on the skin. The shafts of the bones of the legs or of the arms may be enlarged, and hæmaturia or bloody stools may appear.

Pseudo-paraplegia may occur in rickety children from faulty muscular and bony development. It is to be separated from the ordinary paraplegias of childhood by the state of the bones, the presence of knee-jerks, and the absence of local wasting or spasm, but general spasm, or carpo-pedal spasm, is often seen in rickety children.

Not uncommonly a partial paraplegia occurs as a result or sequel of diphtheria. The condition, however, is more ataxic than paraplegic, and Bourges asserts that there is no muscular atrophy such as occurs in true paraplegia due to neuritis, or in some spinal lesions.

When neuritis produces paraplegia it may present symptoms very closely allied to those of acute myelitis, if the symptoms come on suddenly, or of locomotor ataxia; that is, neuritis may cause pseudo-tabes if its onset be slow. The neuritis is always multiple and involves the arms and the body after affecting the legs; there is well-developed anæsthesia (see chapter on Skin), preceded by sensory disturbances and marked muscular and nerve-trunk tenderness; but

there is no girdle-sensation, as there is in myelitis and tabes. There are often trophic changes in the skin in neuritis (see chapter on Skin), but no bedsores as in myelitis. Toxic agents producing a neuritis may sometimes cause a paraplegia of the lower extremities. Da Costa states that malarial neuritis may cause such a symptom, but, as a rule, a toxic neuritis produces loss of power in the arms. Very rarely paraplegia of the lower extremities results from diabetes mellitus, the lesion being in all probability a multiple neuritis.

Monoplegia of a lower extremity may be due to cerebral lesions or to spinal or nerve-trunk lesions. The cerebral lesion producing monoplegia in one leg is very rare, and if it occurs, at any age, indicates a lesion in the convolutions at the upper end of the fissure of Rolando, and the continuation of this area in the paracentral lobule of the marginal convolution. Unlike the paraplegias of infantile cerebral paralysis, monoplegia of the leg very rarely arises from this cause. If there are no signs of cerebral trouble, the presence of a complete leg monoplegia can mean one of several things, namely, a lesion limited to one side of the cord, as, for example, a hemilateral myelitis, hysterical paralysis, in which there will be irregular anæsthesia (see Skin) and the other hysterical signs, or a tumor pressing on the crural nerve in the pelvis, or section of the nerve by injury. Apparent monoplegia may however be due to muscular pain or a painful phlebitis producing muscular fixation.

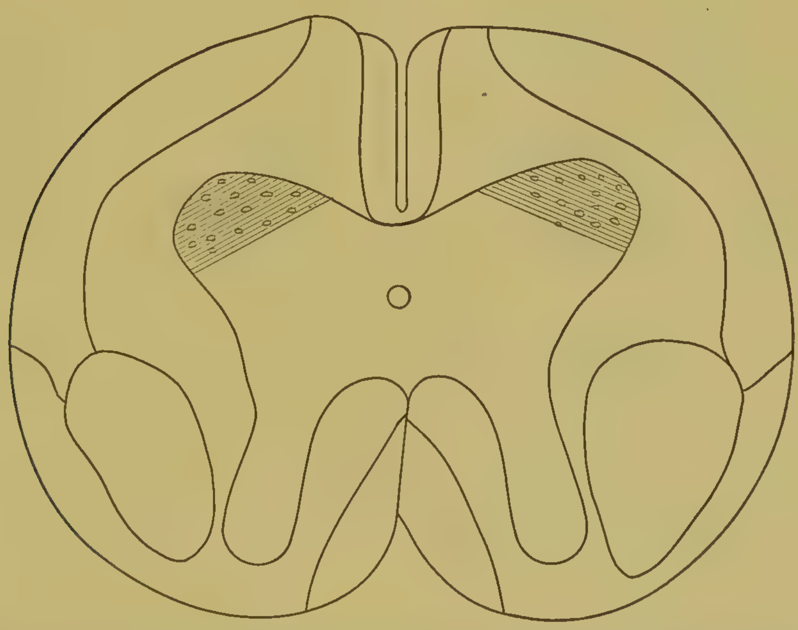
Diagram showing Symptoms in Hemilateral Myelitis.

	Spinal cord.	
Reflexes normal		Reflexes normal.
Band of hyperæsthesia . .		Band of hyperæsthesia.
Band of anæsthesia . . .		Band of anæsthesia.
Reflexes lost		
Motor palsy		Motor power unaffected.
Hyperæsthesia		Anæsthesia.
Reflexes increased.		Reflexes unaffected.
Temperature above the rest of the body		Temperature same as the rest of the body.

If the condition is due to a lesion on one side of the cord, the symptoms are quite characteristic. There is paralysis of all the muscles of the leg which are supplied by the part of the cord affected

or below it. The muscles, the nerve-supply of which comes directly from the affected part, eventually waste and undergo degenerative changes. The most typical symptom of this lesion is, however, the crossed character of the sensory paralysis. That is to say, there is loss of sensation in the opposite limb from that in which motion is lost, and in the limb in which motion is lost there is hyperæsthesia, so that the lightest touch may be very painful. The cause of this is obscure, for the studies of Mott have proved that the sensory tracts in the cord do not decussate on entering it, as has been supposed heretofore. There is, however, a symmetrical band of anæsthesia round the body at the level of the lesions, and a similar band of hyperæsthesia above the lesions. The reflexes of the parts supplied by the diseased area are lost, but those supplied by the area below the lesions are increased as in ordinary myelitis. Very commonly the paralyzed limb is over-warm from vasomotor palsy.

FIG. 38.



Areas involved in acute and chronic poliomyelitis. In children it is sometimes called acute infantile paralysis. Shading shows area of cells in anterior cornua of gray matter which are involved.

Paralysis of certain groups of muscles or a single muscle in the legs is most commonly due to anterior poliomyelitis or neuritis. (Fig. 38.) In poliomyelitis the child will be found to have loss of power in one or both legs in certain muscles (see also Paraplegia), so that there is a dragging of the toe or "foot-drop," the shoe becomes irregularly worn through, being dragged on one edge along the ground, the in-

volved muscles being peculiarly relaxed and flaccid, so that the leg may wobble, to use a crude term. This is sometimes called a "Punchinello leg." There is no tendency to spastic contraction, the reflexes are rapidly lost in the affected part, and the muscles speedily waste and develop the reaction of degeneration. (Fig. 40.) When contractures take place they are not spastic, and are due to healthy muscles being unopposed by the diseased ones. The tem-

FIG. 39.

Case of acute infantile cerebral palsy for comparison with Fig. 40. (SACHS.)

FIG. 40.

Case of infantile spinal palsy: paralysis and atrophy of left leg chiefly. (SACHS.)

perature of the paralyzed part is lower than normal. The history in poliomyelitis is that of sudden onset with fever, vomiting, and restlessness. The two conditions of acute cerebral paralysis and anterior poliomyelitis are so clearly separated in well-marked cases that no error can be made, particularly if the history of the attack be borne in mind, unless it be in the obscure forms of cerebral infantile palsy in the early stages. The above figures show the two

different types of paralysis resulting from these cerebral and spinal diseases in the child. (Figs. 39 and 40.) In acute infantile paralysis of spinal origin the right lower extremity is most frequently affected, after this, a close second, the left leg. Sometimes muscular atrophy may be masked in young children by the abundance of subcutaneous fat. A point of some importance in examining the reflexes is that the presence of knee-jerk should not exclude the diagnosis of poliomyelitis, because the reflex act is only destroyed if the centres which cause this jerk are diseased; that is, if the disease has only affected that part of the cord supplying the foot, a tap on the knee may readily produce a response, whereas if the disease be higher up the reflex will be lost. The chronic anterior poliomyelitis of adult life presents very similar symptoms to the acute form of infancy, but is a very rare disease.

Care must be taken that paralysis of the leg resulting from an injury to the peroneal nerve with resulting neuritis is not mistaken for acute poliomyelitis. The history of an accident, of pain, swelling, and the presence of a bruise aid us in making a diagnosis. If these symptoms occur in an adult, a possible cause is paralysis of the peroneal nerve occurring in the course of tabes. (In connection with this chapter see that on the significance of anæsthesia of the skin.)

Deformities of the Feet and Legs. Much of what has been said in the preceding chapter as to the diseases which produce alterations in the shape of the hand and arm applies equally to the changes from the normal seen in the appearance and movements of the feet and legs. The feet are greatly enlarged symmetrically in acromegaly and in Marie's pulmonary osteo-arthropathy. In the latter disease the enlargement is particularly noticeable because it is the extremities which are chiefly hypertrophied, whereas in acromegaly there is simultaneous enlargement of the shafts of the long bones. It is to be remembered that in both acromegaly and pulmonary osteo-arthropathy the enlargement seems to be due to hypertrophy of all the tissues composing the foot, whereas, on the other hand, in myxœdema the foot, though enlarged, is puffed and swollen in appearance through hypertrophy of the subcutaneous tissues alone. Often the foot appears to be a good deal enlarged as the result of deformity, particularly that which consists in partial displacement of the articular surfaces of the metatarsal and phalangeal bones through the wearing of badly fitting shoes, or joint-troubles of which we shall speak later.

Under the name of "sciopedy" Power has reported a case of congenital symmetrical enlargement of the anterior part of the foot not involving the heel. Any enlargement of the legs associated with this condition, he states, only results from hypertrophy of the muscles resulting from the effort to lift the feet.

The claw-hand spoken of in the chapter on that part of the body is represented by a similar deformity in the foot which may arise from the same causes, in regard to the nervous lesions, and depend upon atrophy of the interossei and other intrinsic muscles of the foot (Fig. 41); but progressive muscular atrophy rarely involves the

FIG. 41.

"Claw-foot" from atrophy of interossei and other intrinsic muscles of foot. (DUCHENNE.)

FIG. 42.

Pes equinus in a boy five years of age, from atrophy of tibialis anticus. (SACHS.)

foot, although it may begin there. When progressive muscular atrophy does begin in the lower extremities, it may fall into the class called progressive neural muscular atrophy, or the peroneal or leg-form of progressive muscular atrophy, affects the leg chiefly, and rarely involves the foot proper first. The extensor muscles of the toes lose their power, the interossei waste, the foot may be flattened or claw-shaped, or, in other instances, any one of the forms of club-foot may develop. If the deformity is bilateral, it is a strong evidence of its being the leg-type of progressive muscular atrophy, and

not due to infantile paralysis. There will probably be a history of heredity in such cases. It must be carefully separated from the pes equinus seen as a result of progressive and acute infantile spinal paralysis involving the tibialis anticus as seen in Fig. 42. The toes are hyper-extended, and the foot is very broad when viewed from side to side at the metatarsal joints. It is stated that this sign is considered characteristic of the early development of the disease in families with the heredity. Sometimes in place of this deformity the foot becomes almost parallel with the tibia in excessive extension, with eversion as the result of shortening of the peroneus longus. (Fig. 43.) In other instances the deformities undergo marked

FIG. 43.

FIG. 44.

Plantar surface exhibiting changes due to contracture of peroneus longus, shortening of transverse diameter, A C, and torsion of foot, D. (SACHS.)

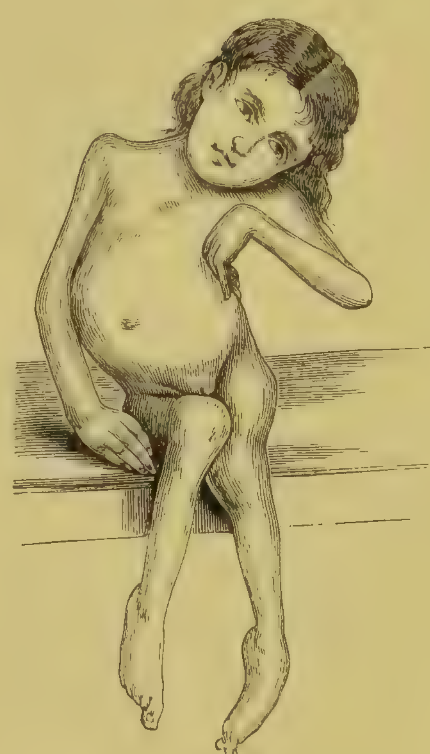
changes as the disease progresses, so that they not only grow worse, but are altered in type. (Figs. 43, 44, 45, and 46.) In distinction from ordinary progressive muscular atrophy this leg-type often has marked disturbance of sensation associated with it. (Dana.) It generally occurs in males. According to Marie, another form of claw-foot is seen in Friedreich's ataxia, there being associated with it club-foot.

Progressive neural muscular atrophy is a rare disease which must

be separated from multiple neuritis by the pain of the latter affection and the fact that neuritis rarely produces double club-foot, and, further, that in neuritis there is no history of heredity. From poliomyelitis we separate it by the fact that in this peroneal type of paralysis the onset is more slow and by the fact that there is a loss of the reflexes in severe poliomyelitis, though they are preserved for a long time in the peroneal type. From Friedreich's ataxia it is separated by the fact that in that disease the reflexes are lost, there is a peculiar unsteadiness in walking, and an absence of electrical changes in the muscles.

FIG. 45.

FIG. 46.



Case of progressive muscular atrophy in a child with a spinal lesion. The three figures (one on p. 107) illustrate the progressive wasting of the muscles and the deformities resulting therefrom. In Fig. 43 hyperextension of the fingers and of the big toe is very striking. (THOMSON and BRUCE.)

The peroneal leg-type of progressive muscular atrophy may so closely resemble the so-called Aran-Duchenne type of progressive muscular atrophy as to defy diagnosis, but as a rule the latter disease affects the arms first and sensation is not involved. (See chapter on Hand and Arm.)

Wasting of the muscles of the inner surface of the foot affecting the big toe, and those on the outer side involving the movements of the little toe, the interossei and the flexor brevis communis, may occur from the neuritis due to locomotor ataxia, and as the plantar aponeurosis retreats the toes are rendered immovably flexed; in other cases in place of flexion we get strong extension, as in this figure. (Fig. 47.)

FIG. 47.

Tabetic foot. (SHINGLETON SMITH.)

A shrivelled undeveloped foot and leg with drawing up and deformity are seen most commonly as the ultimate result of the acute cerebral paralysis of infancy. (Fig. 48.)

Acute cerebral paralysis of infancy is to be separated from spinal paralysis of acute or subacute poliomyelitis anterior of infancy, or the rare poliomyelitis of later life. In these there may be bilateral paralysis, although only one leg and foot are more often involved. Like the paralysis from cerebral disease spinal paralysis comes on suddenly, but there is this marked difference, viz., that in cerebral paralysis there are spastic rigidity and spastic contractures, no atrophy of the muscles, until by disuse or secondary changes in the cord the muscles lose their nutrition, there is marked increase of the reflexes, and the electrical reactions remain normal for a long period of time; whereas in the early stages of acute spinal paralysis there is an entire absence of spastic contraction, the muscles being pecu-

liarly lax, flabby, and lifeless; contractions with resulting deformity only arising from the action of healthy muscles robbed of their natural antagonists. The contractures of the leg which occur in acute infantile paralysis of spinal origin are not spastic, and are often only developed upon intention-movements or by the unopposed healthy muscles.

FIG. 48.

Shrivelled foot of infantile cerebral paralysis. (HIRT.)

A very important form of contracture following paralysis or occurring without it, which is apt to lead to a mistake in diagnosis, is that seen in hysteria. As a rule, the contractures come on in association with paraplegia. Sometimes, however, they affect the arms or an arm. It is a characteristic of these contractures due to hysteria that they set in suddenly, and are always accompanied by such hysterical symptoms as borborygmi, ovarian tenderness, and often areas of anæsthesia. Weir Mitchell has divided these cases into two forms. The first only involves single parts or limited muscle-groups, and, though the contractures may last for years, joint- or muscle-changes do not occur. In the second class, one limb after another is attacked until all means of locomotion, or even moving the trunk, are lost, and the muscles, joints, and areolar tissue undergo organic changes. The reflexes are lost in such cases in the late stages, and the electrical reaction of the muscles is impaired. The diagnosis is to be reached by the sex, the personal history, the history of the illness, the presence of anæsthesia (see chapter on Skin), and hyperæsthesias. Usually the contracture comes on suddenly; it is very rigid and the muscles on both sides of the limb are fixed; that is, the contracture involves antagonistic muscles. Sleep does not always cause a relaxation of hysterical contraction, but ether or chloroform usually does so. (See chapter on Hand and Arm.)

Deformity or distortion of the legs may result from the secondary muscular atrophy following upon chronic inflammation in a joint or

joints. The muscular wasting under these circumstances arises from neuritis, which is associated with the arthritis.

The Joints. The joints of the lower limbs may be swollen from an arthritis arising from many causes, such as locomotor ataxia, hemiplegia, rheumatoid arthritis (arthritis deformans), acute myelitis, cerebro-spinal meningitis, Morvan's disease, septicæmia, or the infectious processes, such as acute articular rheumatism and sepsis.

The most marked alterations in the joints are those produced by advanced locomotor ataxia, and are called arthropathies. Often they are associated with spontaneous fractures of the bones. The knees are most commonly involved, then the ankles and hips. A joint or several joints may become suddenly swollen with or without pain, and without apparent cause until the swelling becomes quite massive. There are then developed osseous hyperplasia and a tendency to dislocation with crepitation on movement, and the ends of the bone become worn away and absorbed. Dislocation and fractures are common, and the bones are atrophied.

In rheumatoid arthritis there is a gradual enlargement of the joints from accumulation of fluid, which in turn is absorbed, leaving the articulating surfaces roughened, uneven, and deformed, but there are no deposits of urate of sodium as in gout, the deformities being due to alterations in the articulating surfaces themselves, and the periarticular development of bone. The disease always remains in the joint originally attacked, although new joints are involved. Pain is often severe, dislocations and fractures are rare, and the small joints are often involved. (See Hand and Arm.)

Rheumatoid arthritis when it progresses to an advanced stage causes great deformity by the locking of the joints through the development of osteophytes. By the destruction of the cartilages, wasting of the muscles, and thickening or contraction of the ligaments it may cause dislocation of all sorts, and false positions. In the great majority of cases it occurs in women between twenty and thirty years of age, but it may develop in early childhood. Pain is severe in some cases, absent in others. The thighs become flexed upon the abdomen, and the leg on the thigh. The number of joints involved varies greatly, but the involvement is generally symmetrical.

Sometimes this disease, which is generally gradual in its onset, becomes very acute, speedily involving many joints, causing swelling of the synovial sheaths and bursæ, and being accompanied by some febrile movement. The suddenness of its onset, the febrile move-

ment, and the pain may cause it to resemble acute articular rheumatism, but the absence of redness in the joints and of the migration of the swelling from one joint to another aids in the differentiation. The arthritis of acute central myelitis is sudden in its onset, generally multiple, and accompanied by the other symptoms of this disease (see Paraplegia and Anæsthesia of the Skin).

The arthritis of cerebro-spinal meningitis is really to be classed as an infectious arthritis, and the presence of the characteristic signs of the disease renders its cause evident. The joints are many of them affected simultaneously with swelling, pain, and serous or purulent effusions. In cases of septic arthritis the joints become swollen and often suppurate, so that the articular surfaces become more or less destroyed. This may occur after infection during the puerperium or in any case of pyæmia. In Morvan's disease or syringomyelia the small joints are usually affected.

The onset of an inflammation in the lower end of the femur or in the upper end of the tibia, producing what, at first glance, seems to be an arthritis and sometimes simultaneously involving other areas near joints, should raise a suspicion of acute osteomyelitis, which is a fatal disease in many cases unless surgical aid comes to the patient. The symptoms consist in boring pain in the part, great tenderness and swelling, and skin soon breaks down as a purulent and offensive discharge makes its way to the surface.

Closely allied to this is the acute epiphysitis of infancy, in which there is suddenly developed a chill followed by great pain and swelling of the joints or their neighborhood. The skin becomes engorged with blood and the joint fills with pus. Care must be taken to separate this condition from rheumatism and the joint swelling sometimes seen after typhoid fever. This state is practically identical with the acute arthritis of childhood.

When arthritis is due to gonorrhœal infection it is generally seen in the knees or ankles, and occurs in men, as a rule. It is an infectious arthritis and lasts very persistently, often attacking at the same time joints so rarely involved by rheumatism as the jaw, the vertebral joints, and the sterno-clavicular articulation. According to the late Dr. Howard, of Montreal, it occurs in five forms:

a. Arthralgic, in which there are wandering pains about the joints, without redness or swelling. These persist for a long time.

b. Rheumatic, in which several joints become affected, just as in subacute articular rheumatism. The fever is slight; the local in-

flammation may fix itself in one joint, but more commonly several become swollen and tender. In this form cerebral and cardiac complications may occur.

c. Acute gonorrhœal arthritis, in which a single articulation becomes suddenly involved. The pain is severe, the swelling extensive, and due chiefly to periarticular œdema. The general fever is not at all proportionate to the intensity of the local signs. The affection usually resolves, though suppuration occasionally supervenes.

d. Chronic hydrarthrosis. This is usually monoarticular, and is particularly apt to involve the knee. It comes on often without pain, redness, or swelling. Formation of pus is rare. It occurred only twice in ninety-six cases tabulated by Nolen.

e. Bursal and synovial form. This attacks chiefly the tendons and their sheaths, and the bursæ and the periosteum. The articulations may not be affected. The bursæ of the patella, the olecranon, and the tendo Achillis are most apt to be involved.

Acute articular rheumatism in the knee or ankle produces swelling of the joint, redness, heat, exquisite tenderness, immobility from pain, swelling of the surrounding tissues. It does not remain for a long period unchanged in one joint, and is a process accompanied by fever.

Although gout is capable of causing deformity in the lower extremities, it has one fact about it which is of practical importance, namely, that it involves the small joints of the foot, while rheumatism attacks the large joints, such as the knee, by preference. Gout involves the feet most commonly, while rheumatism is more frequently seen in the hand, if small joints are affected, and the big toe is the favorite place for gouty manifestation. Aside from the swelling, redness, and exquisite tenderness of gouty joints, all of which symptoms exceed in acuteness similar manifestations in acute rheumatism, there is often an additional and permanent cause of deformity in the chalk-stones which are deposited about the joints, and which are never seen in rheumatism. The history of frequently recurring attacks lasting but a few days, accompanied by enlargement of the veins about the joint and shedding of the skin locally, points, when added to the symptoms named, to a typical case of gout. It may be almost impossible to determine whether a case be one of chronic rheumatism or gout unless chalk-deposits can be found.

Sometimes in chronic lead-poisoning we have developed what is known as plumbic gout owing to the deposition of urate of lead and sodium.

Acute synovitis is generally the result of an injury, is confined to one joint, is often accompanied by a far greater effusion into the joint than is seen in rheumatism, and there is no systemic disturbance. Should a single joint be apparently effaced by an aberrant attack of acute rheumatism or synovitis, the physician should never forget the possibility of its being a gonorrhœal arthritis.

The onset of a multiple arthritis, with which there are headache, chills, intense aching in the bones, joints, and muscles, and a fever rising as high as 106° or 107°, and rarely an erythematous rash, may indicate the presence of dengue. The joints are swollen and painful, and often both the large and small ones are involved. Another arthritis, probably infectious, is sometimes seen in epidemic dysentery and in scarlet fever.

In Schönlein's disease, which is a form of very severe purpura, multiple arthritis, with great pain, and purpuric eruptions occur, and the presence of the subcutaneous exudate with œdema and sloughing of the mucous membrane of the mouth add to the picture. The patient seems very ill, but death rarely follows. Such cases are rare, but the writer saw one in consultation with Dr. Wilson, of Woodbury, New Jersey, in which alarming sloughs of the tonsils and buccal mucous membrane occurred. (See chapter on Skin.)

Very nearly allied to this are the joint-involvements of hemophilia, which in their sudden onset and pain closely resemble rheumatism, particularly as the large joints are commonly involved. The history of the patient being a bleeder, or of his being related to one, may clear up the diagnosis.

Intense swelling of the leg, aside from that due to ordinary œdema, may be due to milk-leg, which is a condition of swelling of the entire limb, generally limited to one side, and seen during the puerperium or after any one of the infectious fevers, such as typhoid. The joints are not particularly affected. On the contrary, the calf of the leg is the part most affected, it being white, firm, hot, but slightly, if at all, œdematous. Pain is excessive, there is entire loss of power in the affected limb, and its temperature is much higher than normal.

If the swelling of the leg is bilateral and pits on pressure, it is practically always the result of anasarca from renal or cardiac dis-

PLATE I.

FIG. 1.

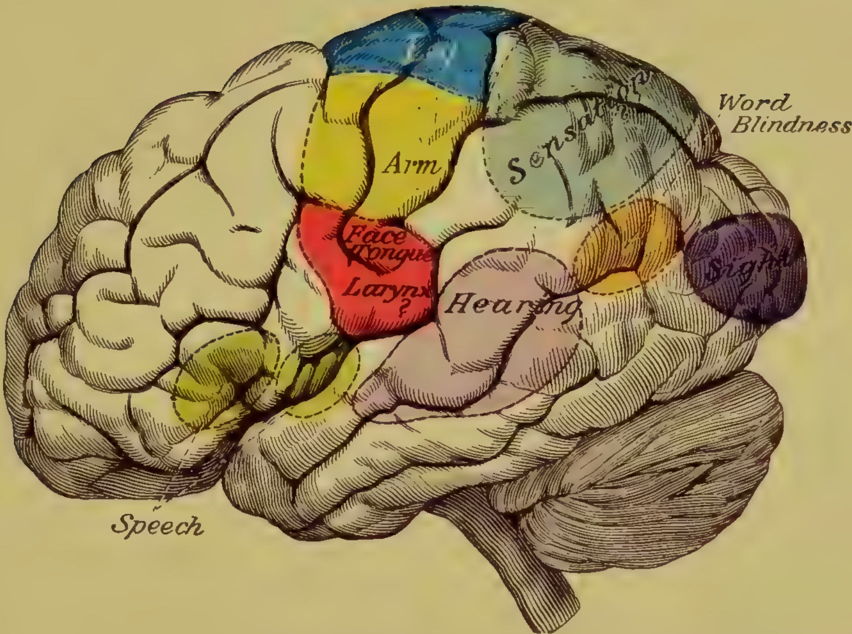


Chart of Localization of Cortical Centres determined on External Surface of Cerebrum. (Gray.)

FIG. 2.

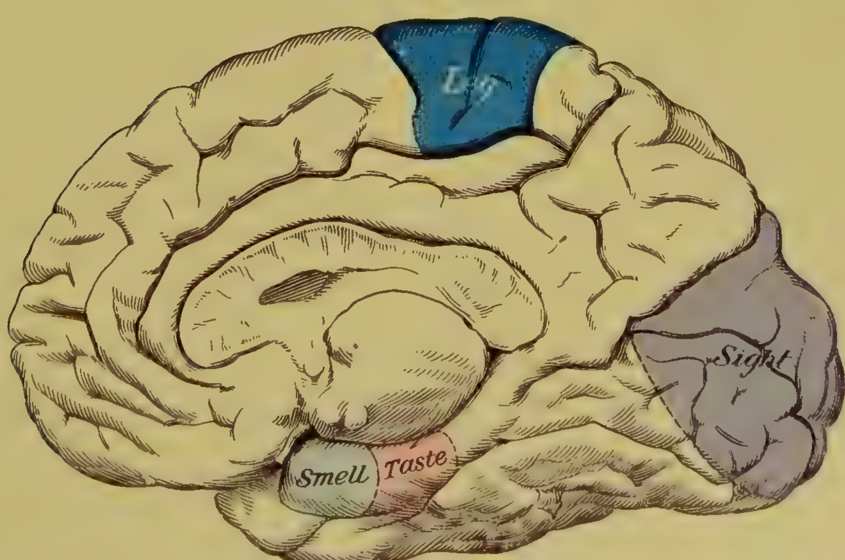


Chart of Localization of Cortical Centres determined on Medial Surface of Cerebrum. (Gray.)

function, and these fibres approximate one another more and more closely in the lower part of the brain until they form a bundle

Fig. 50.

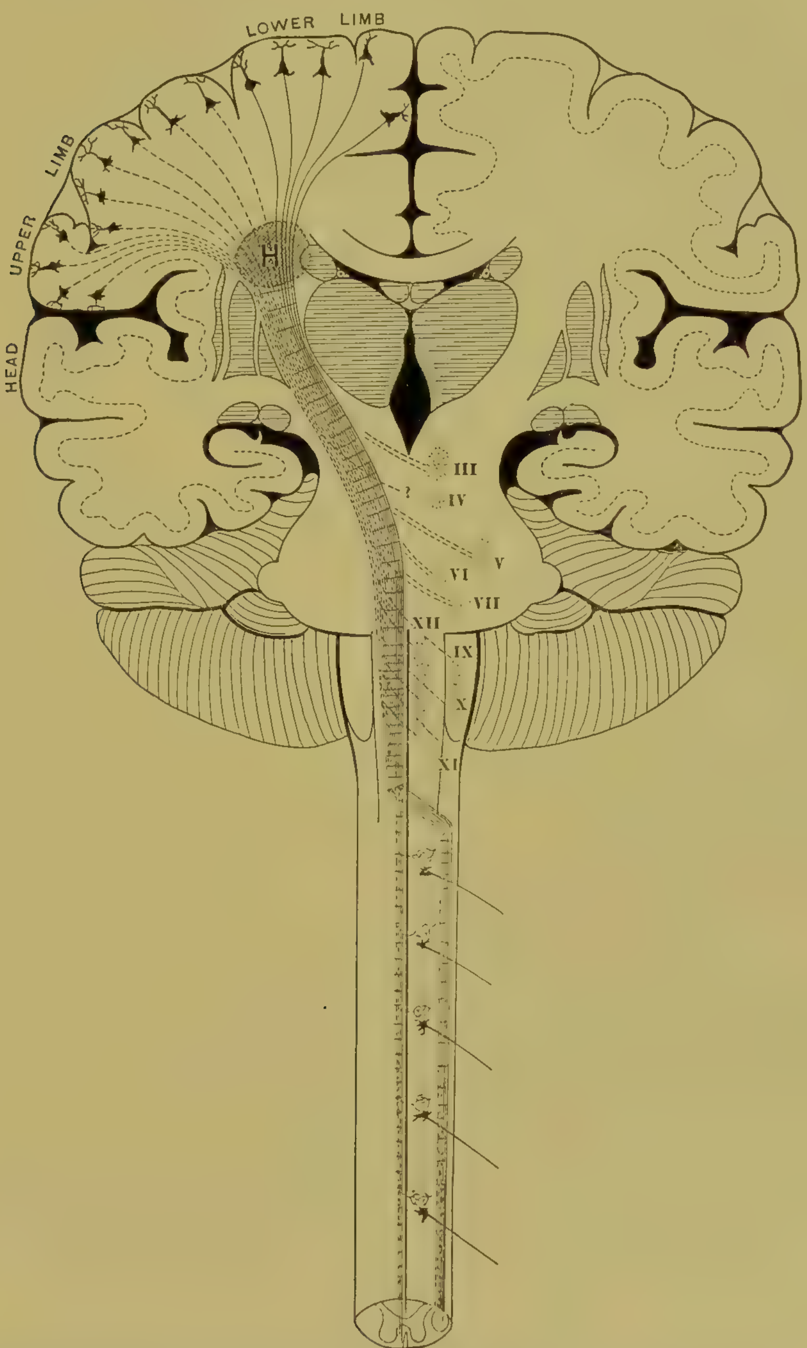


Diagram showing the fibres from the cortex forming the corona radiata, which after they are approximated pass into the internal capsule. It also shows the decussation of the pyramid of the left side, which passes to the right side of the spinal cord, and the direct or uncrossed tract. Finally it also shows secondary degeneration which occurs after cerebral hemorrhage or softening, and which follows the course of the motor tracts into the spinal cord. H. Site of lesion. The continuous lines are fibres going to the legs, the dotted are those going to the arms and motor cranial nerves. (Modified from Van Gehuchten.)

FIG. 51.

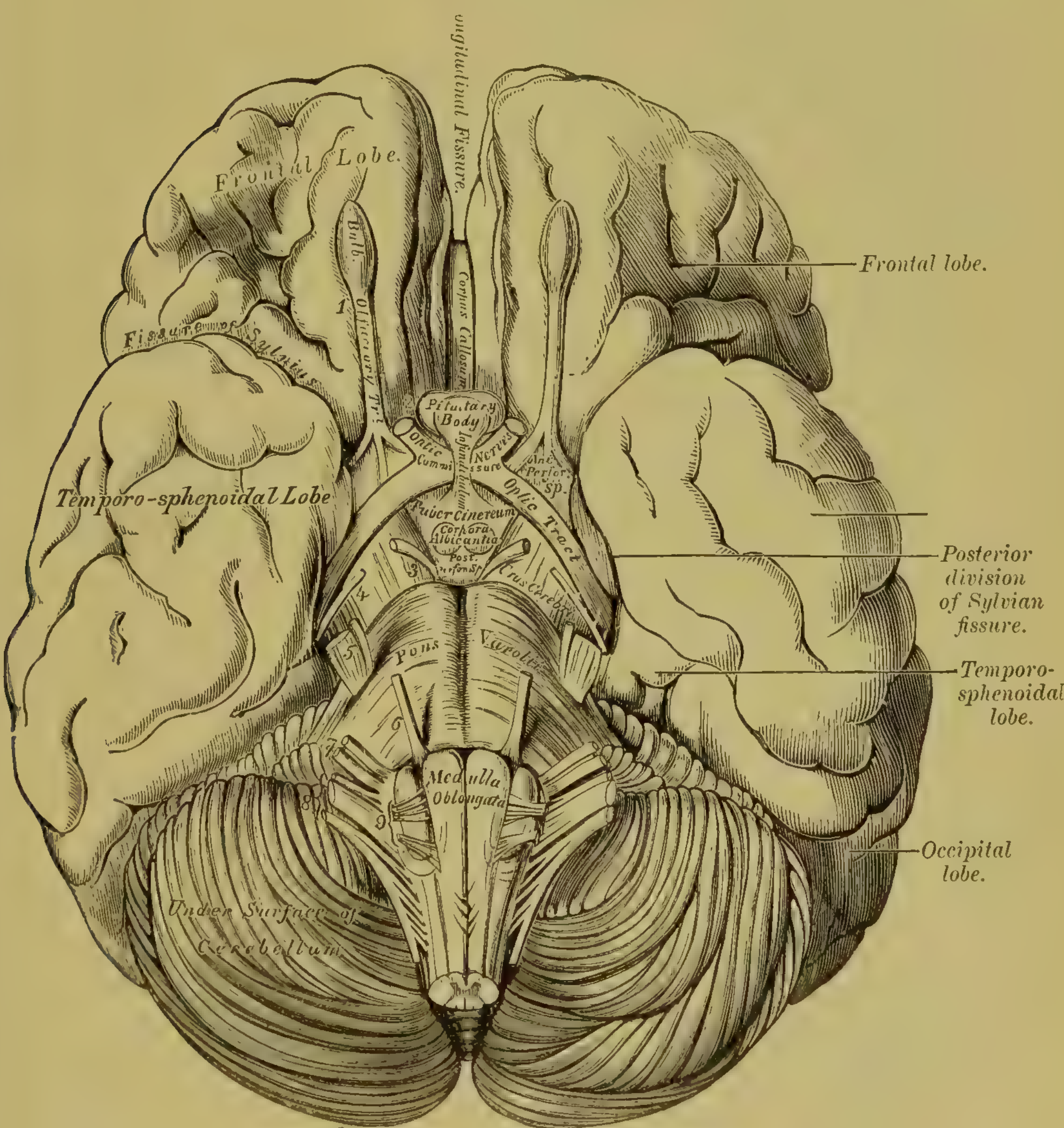
Outline of horizontal section of brain, to show the internal capsule. Natural size. The gray matter of the cortex and claustrum is left unshaded, but that of the corpus striatum and optic thalamus is shaded; *OT*, optic thalamus, showing the median, lateral, and anterior nuclei; *NL*, nucleus lenticularis, showing the putamen large, and the inner division of the globus pallidus very small; *NC*, nucleus caudatus, the large head in front of, and the diminishing tail near the thalamus; *G*, the knee of the internal capsule. From "*Eye*" to "*Digits*" marks the position of the pyramidal tract as a whole, and the several letters indicate broadly the relative positions of the several constituents of the tract, named according to the movements with which they are concerned: Thus *Eye*, movements of the eyes; *Head*, of the head; *Tongue*, of the tongue; *Mouth*, of the mouth; *Shoul*, of the shoulder; *Elbow*, of the elbow; *Digits*, of the hand; *Abdo*, of the abdomen; *Hip*, of the hip; *Knee*, of the knee; *Toes*, of the foot; *S*, the temporo-occipital tract; *oc*, fibres to the occipital lobe; *op*, optic radiation. At this level the fibres of the frontal tract, in the fore limb of the capsule in front of the pyramidal tract, run almost horizontally, parallel with the plane of the section. *cc*, the rostrum of the corpus callosum, *Spl*, the splenium of the same, both cut across horizontally. The thick dark line indicates the boundary of the cavities of the anterior and descending horns of the lateral ventricle and of the third ventricle, the two ventricles being laid open into one by the removal of the velum and choroid plexus, etc. The oval outline in the fore part of this cavity indicates the fornix. Lateral to the nucleus lenticularis are seen in outline the claustrum, the cortex of the island of Reil, and the operculum or convolution overlapping the island of Reil. *P* is inserted to show which is the hind part of the section.

PLATE II.

Diagram showing Course of Motor Fibres from the Cerebrum and Cord to the Periphery. (Flatau.)

(corona radiata). Thus we see in Plate II. how the fibres arising from the middle area of the cortex cerebri pass down through the lenticular nucleus into the knee or angle of what is called the internal capsule. This is a lateral view. In Fig. 50, which also shows the results of a lesion in the capsule, we get an antero-posterior view. These fibres are arranged in such a way that those

FIG. 52.



arising from the lower part of the cortex, as in the face-centre, lie nearest the knee of the capsule, and those highest, furthest from this point. (Fig. 51.) After the motor fibres have passed through the internal capsule, they pass into the crus cerebri of that side,

which (the crus cerebri) connects the hemisphere of the same side with the cerebellum behind it, and the pons and medulla below it. The crura cerebri are two thick cylindrical bundles of white matter which emerge from the anterior border of the pons (Fig. 52), diverge as they pass upward and outward to enter the under part

FIG. 53.

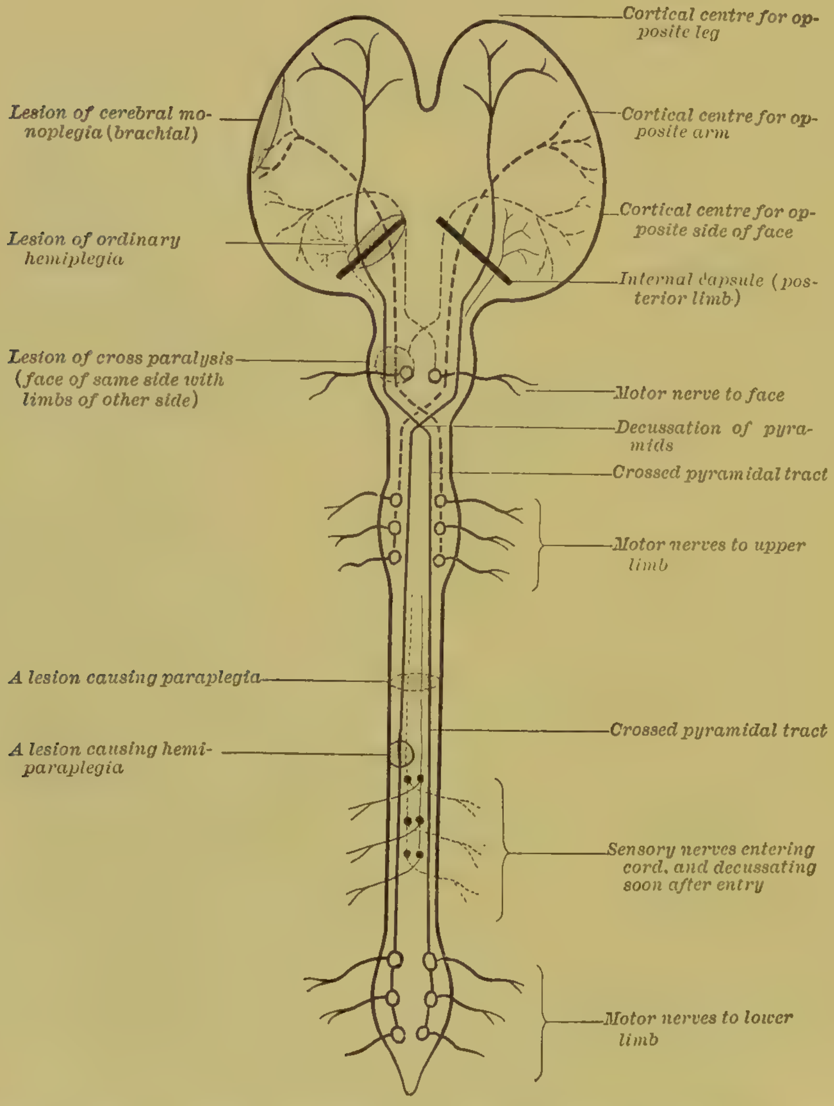
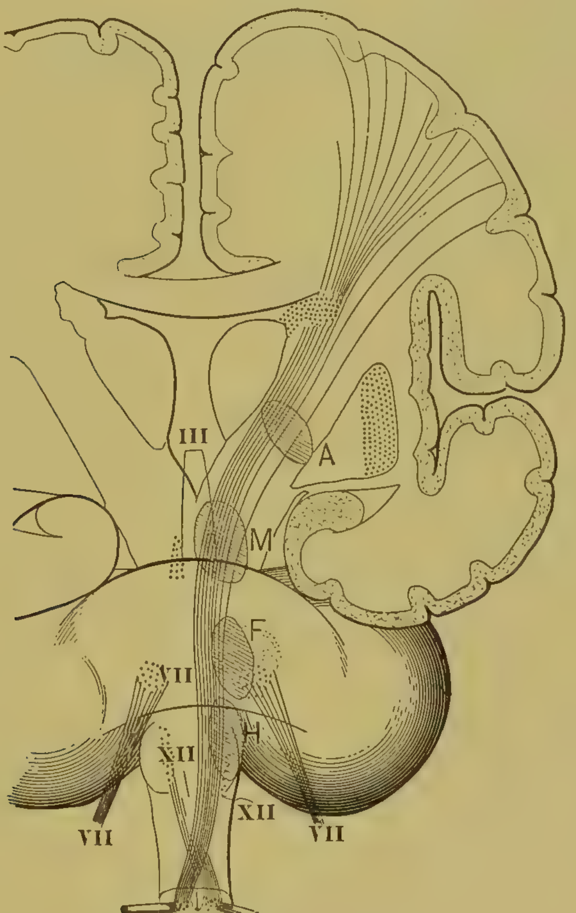


Diagram showing the general arrangement of the motor tract and the effect of lesions at various points.

of each hemisphere, as if stretching out to receive the motor fibres from the internal capsule. From the crura cerebri the motor fibres pass downward into the pons Varolii. Here the fibres which have hitherto travelled together divide into two parts, namely, those from the face and tongue centre, which pass to the opposite side

and become connected with the nuclei of the facial and hypoglossal nerves, which act as minor centres governing the face and tongue, and the fibres for the arm, leg, and trunk of the body which continue on down to the medulla oblongata, where they form the so-called pyramids, and having done so most of the fibres cross to the opposite side of the spinal cord (the crossing of the pyramids), and so form the crossed or lateral pyramidal tracts. (Fig. 50.) A

FIG. 54.



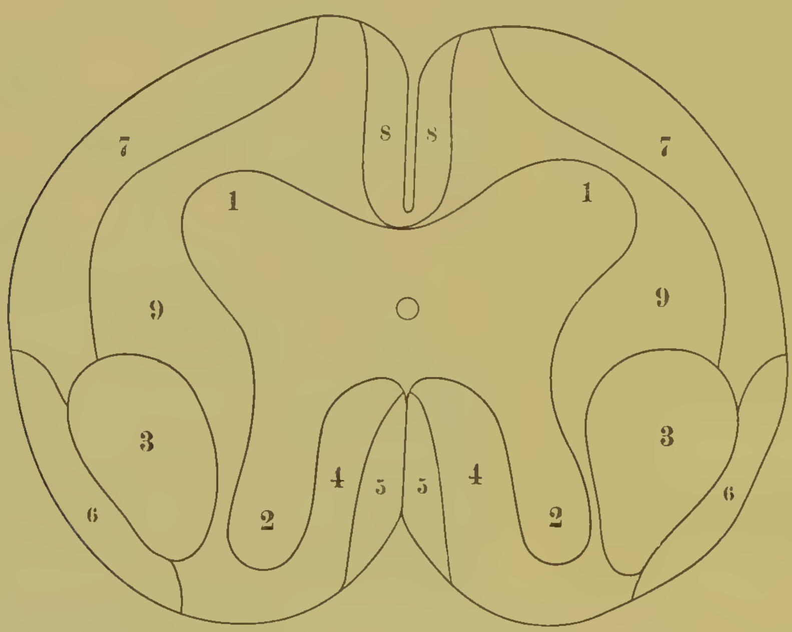
Showing the mechanism of different hemiplegias. A lesion at A causes complete hemiplegia by destroying the motor tract. One at M causes paralysis of third cranial nerve (motor oculi) by destroying its nucleus or root on same side, and paralysis of arm and leg on opposite side. A lesion at F causes facial palsy on same side, hemiplegia on opposite side. In a lesion at H the hypoglossus would be affected on one side, with hemiplegia on the other. (Modified from EDINGER.)

smaller number of fibres, however, pass directly down to the spinal cord from the medulla oblongata, and from what is called the direct or anterior pyramidal tract. Direct, because it does not cross; anterior, because it lies along the edge of the anterior fissure of the

cord; pyramidal, because it comes down from the pyramid. This is sometimes called Türck's column. (Fig. 50.) It is by means of these two tracts in the spinal cord that motor impulses pass down to the nerve-trunks and muscles.

We can understand, therefore, that if a small lesion occurs at the peripheral endings of the corona radiata—that is, on the cerebral cortex—it will only produce a limited paralysis. Thus, as seen in Fig. 53, a clot at the arm-centre would only involve the arm-fibres.

FIG. 55.



Cut showing tracts in spinal cord. 1. Anterior horns of gray matter which contain the cells governing the nutrition of the muscles and give rise to the motor roots. 2. Posterior horns of gray matter which receive the sensory roots. 3. Crossed lateral pyramidal or chief motor tracts from cortex of brain. 4. Columns of Burdach or the outer sensory tracts carrying impulses upward. 5. Columns of Goll or inner sensory tracts carrying impulse upward. Tactile sensibility. 6. Direct cerebellar tract, which carries impulses of a sensory character upward. Tract of muscle-sense. 7. Antero-lateral tracts, which consist of fibres conducting the gray matter of the cord into that of the medulla. They contain anterior nerve-roots and are the channels for reflex effects. There are also tracts in this root for pain and temperature sense. 8. Column of Türck, or direct anterior pyramidal tract, which carries impulses of motion downward. 9. Lateral mixed tracts same as 7.

If, however, the lesion be lower down where the fibres of the corona radiata are getting closer and closer, as, for example, in the internal capsule, then even a small lesion will produce widespread paralysis, since it will involve a large number of fibres running ultimately to widely separated areas in the body, and, if large enough, produce hemiplegia. (Figs. 53, and 54 lesion A.) If the lesion be situated in the pons on one side, it will cause facial paral-

ysis on that side and hemiplegia on the opposite side of the body, because, as shown in the diagram (Figs. 53, and 54 M), it will, under these circumstances, destroy the facial fibres after they have crossed, and the remaining motor fibres before they cross. The various tracts, motor and sensory, in the spinal cord are shown in Fig. 55.

Hemiplegia from hemorrhage is characterized by sudden onset in most cases, by more or less mental disturbance and disorders of motion, sensation, and of the special senses according to the site of the leaking vessel. The skin-reflexes are apt to be markedly decreased and the deep reflexes increased, but the bladder and rectum are not usually paralyzed, although in the first shock of the accident there may be vesical and rectal incontinence. The mental disturbance usually amounts to a rapidly oncoming unconsciousness in hemorrhagic hemiplegia.

FIG. 56.

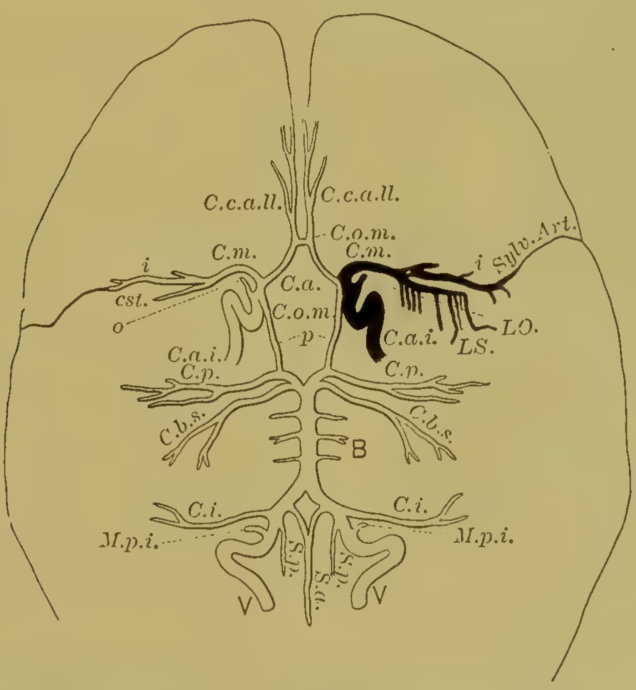


Diagram of the arteries of the base of the brain, showing *LO.*, the lenticular optic, and *LS.*, lenticular striate set of arteries. One of the latter is called the artery of cerebral hemorrhage. *V.A.* Vertebralis. *S.a.* Spinalis anterior. *S.p.* Spinalis posterior. *B.A.* Basilaris with median branches. *C.b.s.* A. Cerebralis superior. *c.i.* Cerebelli inferior. *C.p.* A. Cerebralis posterior (profunda cerebri). *Com. p.* A. Communicantes posteriores. *C.a.i.* Carotis interna. *o.* A. Ophthalmica. *C.m.* A. Cerebralis media (A. fossæ Sylvii). *i.* A. Insularis. *cst.* A. Corp. striati. *C.a.* A. Cerebralis anterior. *Com.* A. Communicans anterior. *C.c.a.ll.* A. Corp. callosi.

The question of the location of the lesion is very important. In the great majority of cases it is situated above the point at which the decussation of the motor fibres takes place in the medulla, and

is, therefore, on the opposite side of the body from that on which the hemiplegia exists. If, however, the lesion be below the decussation, the paralysis and lesion are on the same side, as just described. The most common site for the lesion in hemiplegia is in the knee or posterior limb of the internal capsule, owing to the fact that the middle cerebral artery in one of its lenticulo-striate branches perforates the internal capsule, and ends in the caudate nucleus, and this artery is so commonly ruptured that Charcot has called it the "artery of cerebral hemorrhage." (Fig. 56.) If the hemorrhage does not involve the posterior third of the internal capsule, there are no sensory symptoms associated with the motor loss, but the paralysis will be practically universal on that side, involving the leg and arm, and the lower part of the face, so that the mouth is drawn toward the healthy side. (Explained by Fig. 51.) The symptoms associated with hemiplegia due to this cause often become very severe because the hemorrhage is so profuse that the lateral ventricles become filled with blood, and from them the blood passes to the third, and from there to the fourth ventricle, where, by pressure on the vital centres, it speedily produces death. In such cases deep unconsciousness, stertorous breathing, a slow, full pulse, and a flushed skin, becoming somewhat cyanotic, may be present. Recovery never occurs, for the secondary inflammation, or softening, following the outflow of blood produces fatal results, even if the patient survives for some days.

In cases in which the hemorrhage is very limited consciousness may be lost for only a brief period, and at most there may be mental confusion. Often in mild cases there is a slight return of power in the affected side within a few days, and the temperature of the affected part, which has been raised, approaches the normal. Finally, after six to eight weeks the dominant symptoms consist in partial loss of power of the arm and leg, and the facial paralysis has perhaps entirely disappeared, although the tongue when protruded may tend to go over to one side. If the case does not pass to such favorable results, instead of recovery of power at this time there are developed contractions and secondary rigidity from degenerative processes extending to the pyramidal tracts. (See Fig. 50.) Hitzig has shown that these conditions are apt to be least marked in the morning. Wasting of the paralyzed muscles only ensues from the disuse, and not from trophic change.

When the case is not of the very severe type which causes death in a few hours, and yet the lesions are such that recovery is not going to

take place, the patient at the third or fourth day becomes unconscious a second time, his temperature rises, he mutters, and grows restless, finally becomes comatose, then develops respiratory failure, or a hypostatic congestion of the lungs, and dies.

When a patient is seized with headache, dizziness, vertigo, and vomiting, and rapidly oncoming hemiplegia and hemianæsthesia, attended at first with no loss of consciousness, but in a day by unconsciousness and coma, he is suffering from what has been called "ingravescent apoplexy." The hemorrhage, under these circumstances, begins in the knee of the internal capsule, proceeds backward till it involves the sensory fibres in the internal capsule, and, finally, breaks into the lateral ventricle, soon after which death ensues.

When a hemiplegia is followed by rigidity very early, with sensory involvement and convulsions, the lesion is probably cortical, or, more correctly, is secondarily cortical to a deeper hemorrhage, and spreads over the centres for the face, arm, and leg. Most commonly, however, cortical hemorrhages are due to injuries, or they may arise from unprovoked vascular rupture. In any case, they are usually ushered in by convulsions.

Where, on the other hand, there is paralysis of the arm, trunk, and leg on one side, with facial paralysis and anæsthesia on the opposite side of a well-marked type, associated with early rigidity of the paralyzed side, conjugate deviation of the eyeballs, very marked rise in bodily temperature, a contracted pupil, and convulsions, with difficulty in swallowing and in speech, the lesion is to be found in the pons Varolii on the side opposite the paralysis. This is due to the fact that the injury is below the decussation of the facial nerve. (Figs. 53 and 54.) If both sides of the face are paralyzed with hemiplegia elsewhere, the lesion is in the pons where the facial fibres cross. Pons paralysis is nearly always associated with giddiness, vomiting, conjugate spasm with nystagmus, albuminuria, glycosuria, and marked disturbances in the respiration and heart. Pontile hemorrhages are, however, very rare, and usually are rapidly fatal.

Finally, if there is hemiplegia of the lower part of the face, arm, and leg, and in addition paralysis of the upper part of the face, and ptosis from paralysis of the facial and oculo-motor nerves on the opposite side, and in association impaired sensibility and vasomotor changes in the limbs, the lesion is probably in the crus cerebri on the side of the upper facial paralysis—that is, on the same side as the ptosis; but this is only true if the two paralyses have been

simultaneous in occurrence, as it is possible for one lesion in one place to produce paralysis of the face and another elsewhere to produce the hemiplegia (see Ptosis in chapters on Face and on the Eye).

If in the development of symptoms of cerebral hemorrhage there be little hemiplegia and temporary unconsciousness, followed in some hours by a sudden aggravation of the symptoms, it may be that in the beginning of the attack the lesion has been in the frontal lobes, but has gradually extended backward until it has ruptured into the lateral ventricle. So, too, a hemorrhage into the occipital lobe or the posterior part of the parietal lobe is rarely marked by much hemiplegia, and, if present, the leg is more paralyzed than the arm. The characteristic symptom, however, is well-marked hemianæsthesia (see chapter on Skin), and hemianopsia (see chapter on Eye). Generally, however, such changes result from a thrombosis.

When there is developed, in cases of hemiplegia, aphasia or disordered speech, there is probably a lesion in the neighborhood of the third frontal convolution, or the island of Reil (see chapter on Speech).

Hemiplegia may be due to cerebellar hemorrhage, in which case there are loss of consciousness deepening into profound coma, contracted pupils, vomiting in many of the cases, and finally death when hemorrhage breaks into the lateral ventricle. The diagnosis of cerebellar hemorrhage is very difficult.

Of the irregular forms of hemiplegia there are several. Sometimes the leg is from the beginning more affected than the arm, and remains paralyzed long after the face and arm have recovered. The leg may become rigid and distorted by contractures, and there will often be found present marked anæsthesia of the skin of the paralyzed leg and arm, with hemianopsia and aphasia. Such symptoms indicate a lesion of comparatively small size involving the leg-fibres and some of the sensory fibres in the internal capsule, and results from rupture of the lenticulo-optic artery. When the arm suffers most the symptoms just described as in the leg are seen in it, and motor aphasia, if the lesion is on the right side, is often very marked, as is also facial paralysis. This is supposed to be due to the anterior frontal artery, a branch of the anterior cerebral artery, becoming diseased.

When post-hemiplegic chorea attacks the paralyzed limbs there is often a focal lesion in the posterior extremity of the internal capsule.

The symptoms we have just detailed may also arise, as we have

already said, from embolism or thrombosis of the cerebral vessels as well as from hemorrhage from them. How are we to separate the hemiplegias of hemorrhage and occlusion? In many cases this is impossible, but there are some differential points which may aid us. In the first place, thrombosis is a condition of advanced age, while hemorrhage may occur at any time from thirty years of age on. The presence of hemiplegia in a young man, therefore, is probably not due to a thrombosis. Again, hemorrhage occurs often after exertion or the drinking of stimulants, and occurs rarely in sleep, whereas thrombosis not rarely comes on under these circumstances, and often develops during the night, so that the patient awakes paralyzed, and a patient may have both thrombosis and apoplexy. In hemorrhage, consciousness is generally lost, whereas in thrombosis it is often only dimmed. Vomiting and contracted pupils from pressure on the lower centres indicate hemorrhage, while their absence may point to thrombosis. Finally, the general systemic shock and febrile movement are apt to be greater in hemorrhage than in thrombosis. The history of syphilitic infection, producing an endarteritis, also points to thrombosis, although hemorrhage may arise from this cause also.

The diagnosis of embolism producing hemiplegia from the paralysis due to hemorrhage is always more or less difficult, but the presence of chronic or ulcerative endocarditis or their results, or other cause for the formation of emboli, aid the diagnosis. Where the cause is embolism the onset is sudden, whereas in thrombosis it is sometimes more gradual. The paralysis from embolism is more commonly on the right side of the body, owing to the fact that it is more easy for an embolus to pass into the left middle cerebral artery than into the right.

Spastic hemiplegia may be due to cerebral tumor, and is often associated with convulsions, particularly if the growth be cortical. Very often the paralysis of cerebral tumor will be found, from the history, to have come on gradually. Thus, the history may be that at first the side of the face has been paralyzed, then the arm and then the leg, and that the complete loss of power has not been sudden but gradual in the part affected, or that a convulsion has left that side, which was previously only impaired in strength, totally paralyzed.

Hemiplegia also comes on as a result of cerebral syphilis, and, aside from a history of specific infection and response to specific medication, presents few characteristic signs. The presence of intense

headache, convulsions of an epileptiform type, and the fact that the paralysis occurs in some cases in early youth, point to its origin.

Another cause of hemiplegia is diffuse cerebral sclerosis of one hemisphere (not multiple sclerosis), in which the most constant symptoms are, in addition to the paralysis, evidences of motor irritation, such as epileptoid convulsions of a bilateral or unilateral character, rhythmical or arhythmical twitchings of the muscles like chorea, and dementia.

Hemiplegia may also arise from hæmatoma of the dura mater. The symptoms, aside from the paralysis, are headache, stupor, irregular pulse, vomiting, and contracted pupils, or, in other words, symptoms of cerebral compression. Sometimes twitchings of the paralyzed side occur, and if the clot be near the third frontal convolution or the island of Reil, aphasia may be present. Sensation is usually not involved. The diagnosis of this form from that due to hemorrhage is often impossible.

Hemiplegia arising from acute infantile cerebral paralysis has many of the distinctive features already described when discussing the paraplegia due to this diseased state. The age of the patient, the occurrence of epileptiform convulsions and of athetosis in the affected parts, and the patient's history are the important points to be recalled in making a diagnosis. The lesion is always due to a cerebral hemorrhage or to embolic softening.

When hemiplegia occurs in locomotor ataxia, it depends not upon the disease, but upon a complicating hemorrhage, embolism, or thrombosis.

A slowly developed hemiplegia sometimes results from multiple sclerosis, the pathological process involving the side of the pons and spinal cord, but the intention-tremor, the peculiar speech, the nystagmus, and the very excessive reflexes aid us in the diagnosis of this cause of the loss of power.

A form of hemiplegia which is often very misleading is that occurring in general paralysis of the insane or paretic dementia. In this disease the patient often has attacks of vertigo, unconsciousness, and more or less marked hemiplegia or monoplegia, sometimes with aphasia if the right side is paralyzed. This form is also liable to be wrongly diagnosed by reason of the epileptiform convulsions, which frequently occur, and which in connection with the paralysis give the impression in the first attack that there is a hemorrhage of the cerebral cortex. The presence of the altered disposition of the

patient, the loss of memory and intelligence, the peculiar stumbling speech, and the curious changes in the handwriting are some of the symptoms which complete the diagnostic picture.

Hemiplegia sometimes comes on in purulent meningitis. The history of a head-injury or of a pyæmic or infective process, the cerebral symptoms, the stiffness of the back of the neck, the impairment of the normal movements of the eyeball, and the optic neuritis, associated with the convulsions, make the diagnosis possible.

A very rare form of paralysis, in which the arm on one side and the leg on the other side are involved, is due to a bulbar lesion just where the decussation of the pyramids takes place. This is called crossed paralysis, and is due to cutting off of one set of fibres before they cross, and the others after they have crossed. (See chapters on Hand and Arm, Feet and Legs, and Face and Head for further information as to crossed paralysis.)

CHAPTER V.

THE TONGUE, MOUTH, AND PHARYNX.

The general appearance of the tongue—Its coating—Its appearance in poisoning—Fissures and ulcers of the tongue—Eruptions on the tongue—Atrophy and hypertrophy of the tongue—Paralysis—Tremor and spasm of the tongue.

THE appearance of the tongue is recognized as indicative of the general condition of the patient, and is a valuable diagnostic aid in many diseases other than those associated with disorder of the gastro-intestinal mucous membrane. In examining this organ the physician should take note of the condition of its surface, its shape as it lies in the mouth or is protruded, and the character of its movements. He should also see that it is well protruded, and examine the back of it more than the tip, as the latter is the part giving the least information.

Before discussing the precise appearance of the tongue in the various disorders in which it becomes altered in appearance, it is well to remember that its surface is covered by mucous membrane, which differs in various parts. The epithelium is scaly and rests upon the corium or mucosa. The mucosa also supports many papillæ which are thickly distributed over the anterior two-thirds of the tongue on its upper surface. These papillæ give the peculiar roughness which is so characteristic of this surface, and occur in three forms, namely, the circumvallate or large papillæ, the fungiform or mediate, and the filiform. The circumvallate are only eight or twelve in number, and are arranged at the back of the tongue in the shape of the letter V, with the point toward the root of the organ. The fungiform papillæ are scattered freely over the tongue, mostly at the sides and tip, and appear as deep-red eminences, the bases of which are smaller than their free extremities. Their epithelial covering is very thin. The filiform papillæ, which cover the anterior surface of the tongue, are very minute, and arranged in lines corresponding in direction with the two rows of the circumvallate papillæ. From their apices project many fine filiform processes, which are of a whitish

tint owing to the density of the epithelium of which they are composed. There are, in addition, many simple papillæ which cover the surface between the peculiar ones already described. It is the fungiform papillæ which are seen most commonly in cases of disease, for they become large and prominent, and because of their red color show through the coating as red dots.

The appearance of the surface of the tongue varies greatly even in health according to the condition of its mucous membrane and the epithelium covering it. The most common alterations in its appearance are due to mere superficial coatings or fur, which consist of dead epithelial cells, micro-organisms of many kinds, and abnormally shaped living epithelium. Small particles of food may also be present. Butlin believes that the coating is chiefly due to micro-organisms. The question as to how characteristic of a particular disease any one coating or fur may be has been warmly discussed. Some have gone so far as to assert that the coating of the tongue is not indicative of any state in particular, while others, of whom the author is one, are convinced that while an absolute diagnosis of disease in other organs cannot be based upon the appearance of the tongue great aid can be gained by its study.

There are, however, very few conditions of the coating of the tongue which are pathognomonic of any one disease, since the coating is produced by the local conditions of the mouth rather than by the disease itself.

Taking up for consideration the various forms of coating, we find that the area at the base between the circumvallate papillæ is always somewhat coated even in the best of health, and that in disease the heaviest coating is generally found in this region, while the tip and sides, even in those diseases in which the coating is heaviest, are generally fairly clear. This is in part due to the character of the epithelium in different parts, and to the fact that the tip and sides are generally scraped clean by the movements of the tongue. Further, it should be remembered that the development of coating, aside from digestive derangements, depends chiefly on three factors: first, immobility of the tongue, so that it is kept clean by rubbing; second, mouth-breathing, whereby the surface becomes dry and less easily cleansed; and, third, fever, which not only dries the surface of the tongue by mouth-breathing, but interferes with salivary secretion. Additional local causes are a decayed or ragged tooth or follicular tonsillitis, which infects the lingual epithelium, lack of cleanliness,

and habits, such as smoking. In the last class of patients a heavily coated tongue in the morning is very common.

The tongue of the typhoid state, and of typhoid fever in particular, is quite characteristic, because the prolonged illness, the great exhaustion, and the general apathy of the patient all conspire to produce a peculiar coating on this organ. Early in the disease the surface of the tongue may be more or less foul, resembling the tongue we shall describe in biliousness, in that the back part is coated evenly and with a paste, but very soon a characteristic sign appears, namely, that the tip of the tongue and its edges become red, and the coating becomes most marked on each side of the median fissure, which increases in depth from before backward. The tongue also becomes narrow instead of broad and flabby, as it is in biliousness, and is drier. If the attack be mild, this condition may remain till convalescence is established; but if the disease runs a severe course, the coating becomes very heavy, more dry, rough and brown from exposure to air and medicine. The furred appearance becomes almost shaggy at the back portion, and the drying proceeds until the underlying epithelial layer is cracked and fissured, so that tiny exudations of blood add to the lingual discoloration. The reddened edges become dusky in hue, and may be cracked and fissured also. (Fig. 1, Plate III.) The tongue is very slowly protruded on request, partly from mental apathy, partly from feebleness and because its surface is so stiffened that to move it is difficult. It is equally slowly withdrawn for similar reasons, and while protruded is often markedly tremulous. Toward the close of the attack the tongue cleans off through exfoliation of the dead epithelial accumulation, and this is a favorable or unfavorable sign according to whether the remaining surface is red and moist or dusky and dry. Sometimes these characteristic coatings do not appear, the tongue being brown and rough all through the disease.

A small triangular patch devoid of coating is often seen at the tip of the tongue in relapsing fever.

In biliousness the tongue is coated almost uniformly by a whitish-yellow, pasty coat, extending from back to tip and side to side. The tongue is broad and flabby, and sometimes indented by the teeth, while the breath is foul and heavy. (Fig. 2, Plate III.) A similar tongue is seen in severe tonsillitis, except that it seems even more foul and less yellow in tint. Similarly in jaundice of the acute catarrhal type we have a coating still more yellow in some cases, because, as

Fothergill asserts, the coat has been stained by the tauro-cholic acid eliminated by the salivary glands. The circumvallate papillæ are often prominent and stand above the coating, which is easily removed on scraping.

A broad, white, heavily coated moist tongue is often seen in acute articular rheumatism, becoming dry if the fever is high and the attack prolonged.

The white tongue of persons who take large amounts of milk is generally not smooth and pasty, but rather rough in appearance. If the tongue be suffering from an attack of thrush (saccharomyces albicans), the white coating will consist of irregular white masses of the growth, which, if in great number, often coalesce and make a fairly even surface. The soreness of the mouth, the local heat, the salivation, and the age of the person—generally a young child, render the diagnosis easy.

A grayish diphtheritic-looking coating of the tongue occurring in adults may be due to the growth of various forms of mycoses. Thus, a fine network of leptothrix in threads and tufts often spreads over the tongue, particularly in the region of the circumvallate papillæ. The growth may be quite dark in color, but it is separated from the exudate of diphtheria by microscopic study and the absence of systemic disturbance.

Sometimes on examining the tongue of a child we find that it is broad and flabby and covered by a gray coating which is smooth and fairly moist. Scattered throughout this coating are patches in which the coating and epithelium have been shed, leaving red spots with sharply defined edges, which spots are said to be "worm-eaten" in their appearance; that is, to have the irregular outline of the marks on a worm-eaten leaf. In these areas are to be seen enlarged and reddened fungiform papillæ. Such a tongue is typical of what has been called, by Eustace Smith, "mucous disease," a condition in which there exists a more or less marked chronic catarrhal process in all the mucous membrane. (Fig. 3, Plate III.) If, on the other hand, there is a comparatively light coating dotted irregularly by bright-red spots, which are not raised above the surface, but are very numerous, and the patient is a child, the diagnosis may be made of acute or subacute gastric catarrh. (Fig. 4, Plate III.)

The so-called strawberry tongue is one in which the organ is covered by a thick whitish coat, through which project the fungiform

papillæ, which have been deprived of their epithelial covering, and being swollen or enlarged stand out prominently. This appearance of the tongue is seen commonly in scarlet fever, but is not, as has been thought, pathognomonic of that disease. The fungiform papillæ in the strawberry tongue of scarlet fever are, however, particularly prominent and erect.

When the tongue is excessively furred or rough in appearance, the coating is due to abnormally long and projecting papillæ covered by an excess of living and dead epithelial cells; it may denote grave disease of the viscera, but in rare instances possesses no diagnostic importance, unless coupled with other symptoms. This tongue is sometimes seen in scrofulous children in whom strumous manifestations are marked.

Should the tongue be denuded not only of coating, but, in addition, of its normal epithelium, so that it appears dry, hard and harsh to the touch, it denotes, as a rule, grave and advanced disease of an exhausting nature, such as renal, hepatic, or gastric disorder about to cause the death of the patient. Sometimes this condition is seen in advanced phthisis or gastric carcinoma, and is of evil omen. When it is bereft of epithelium, beefy and red-looking, elongated and narrowed, and shows a peculiar roundness when protruded, severe visceral disease of the abdominal organs, such as dysentery, or hepatic abscess, or carcinoma, will often be found, or, in some cases, this condition develops to add to the discomfort of cases of advanced pulmonary tuberculosis or acute peritonitis. This tongue is sometimes called the "parrot tongue."

In this connection the point should be noted that dryness of the tongue in the presence of grave disease is always an evil omen, and returning moisture of the tongue a favorable one.

Unilateral coating of the tongue may be due to a decayed or ragged tooth, or to hemiplegia, which prevents that side of the tongue from being cleaned through movement. Hillow and Fairlie Clark both assert that morbid conditions of the second division of the trifacial nerve cause unilateral coating, and that abnormalities of the third division do not produce these changes as we would expect.

The coating of the tongue is often so stained by extraneous substances as to be entirely changed in appearance. If the coating be black, the color may be due to the ingestion of iron, of bismuth, charcoal, ink, or blackberries, mulberries, cherries, or red wine. In very rare cases it is black, not from the growth of a fungus, as

has been thought, but from overgrowth of the epithelium with the deposit of a black pigment of unknown origin. Usually this brownish-black discoloration is confined to the middle of the tongue. The affected surface is often rough, due to the enlarged papillæ, and the edges of the spot are less black than the centre. In professional tea-tasters the tongue may be orange tinted.

The coating may be stained brown from the chewing of tobacco, licorice, nuts, and prunes, or chocolate, and yellow from the ingestion of laudanum or rhubarb.

The color of the tongue itself, aside from discoloration of its epithelium, is an important diagnostic aid. It is exceedingly pale in all forms of anæmia, particularly those due to lack of hæmoglobin, such as chlorosis or acute anæmia from hemorrhage, and in pernicious anæmia, when well advanced, it has a remarkable pallor. It is livid and cyanotic in cases of pulmonary disease interfering with oxidation of the blood, or in cardiac disease with similar difficulty.

Purple spots, which may be almost black, may be present in Addison's disease. Sometimes they are bluish-black, and always well defined and even with the surface. Very rarely the tongue is discolored by infarcts, blood-stains, and bruises. When the tongue has its edges dotted with yellowish patches of a slightly elevated character the condition is xanthelasma, and the liver will often be found to be disordered.

In cases of poisoning by corrosive sublimate the tongue presents a most characteristic appearance, for it is white and shrivelled, and the papillæ at the base are unusually large.

When sulphuric acid has been swallowed the tongue has a parchment-like appearance, is at first white and then gray or brownish-gray, and finally covered by a black slough, which as it separates leaves a swollen excoriated patch. In nitric- and chromic-acid-poisoning the tongue is shrivelled and lemon-yellow in color, as it is when hydrochloric acid has been swallowed. The tongue of carbolic-acid-poisoning is very characteristic indeed, for it is shrivelled and puckered into folds. The spots where the acid has touched are brownish if impure acid has been swallowed, or white if the pure acid has been taken. In the course of a few hours this spot becomes surrounded by a red zone, and finally becomes dark brown or black in the centre. After oxalic acid is taken the tongue may be covered by a thick white coat and looks as if it had been scalded. Caustic potash and soda soften the mucous membrane, so that it is pulpy and easily detached, and

looks pearly red or yellow in hue. When ammonia is swallowed the color is white, but superficial œdema may make it pearly in appearance, and acid nitrate of mercury renders it very red. Cantharidal poisoning produces large lingual blisters and sores.

Aside from the coating and the color of the tongue, its surface should be examined to discover fissures, cracks, ulcers, sloughs, and swellings. The tongue is often seen to be superficially and irregularly fissured in old persons, particularly in those who have used large quantities of strong alcoholic drinks or strong tea, or who have chewed tobacco incessantly for many years. The fissures cross each other in every direction, although the central fissure which runs longitudinally is generally deepest and longest. If the furrows are very deep, they may indicate the early stages of what Wunderlich has called dissecting-glossitis, which in turn may be due to syphilis,[1] although, as a rule, the fissures of the tongue due to syphilis are deepest at the edges of the organ, and are due to pressure by and from irritation from the teeth or to ulceration, and subsequent cicatrization of small syphilitic nodules or gummata. The cervical glands are rarely involved in such cases. If only one ulcer is present, it may be chancre, which will have the peculiar Hunterian hard base, and, in such a case, the cervical glands will probably be enlarged. An epithelioma may also have an indurated base with secondary glandular enlargement. Lingual ulcers may also be present as the mucous patches of syphilis, or be due to wounds from the teeth, a broken pipe-stem, or a fork. When these become chronic their separation from those due to syphilis and tuberculosis is practically impossible on superficial examination. Sometimes an ulcer of the tongue is due to epithelioma; but if this is the case, the patient will probably be past thirty years of age. As deep syphilitic ulcers heal sclerosis of the tongue may develop.

Multiple ulceration of the tongue may be due to tubercular disease, which is very rarely primary, but rather secondary to its presence elsewhere. The sores are often stellate in shape, and there is always swelling of the cervical lymphatics, whereas in multiple syphilitic ulceration of the tongue the glands generally escape. The diagnosis between tubercular ulcer and that due to epithelioma is more difficult, since in both diseases the cervical glands are involved. Both are more common in men than in women. The

[1] This is denied by Demarquay and doubted by Butlin.

age of the patient, the presence of tubercular disease elsewhere, and the absence of induration point to tubercle. The tuberculous ulcer is not surrounded by much inflammation, is covered by grayish purulent mucus, and may contain bacilli of tubercle, and is often associated with tubercular nodules, which have not broken down.

Ulcers of the tongue may also be due very, very rarely to lupus. A very similar tongue is seen in a tropical disease with intestinal disorder called by Thin "psilosis." An herpetic eruption appears on the tongue, which leaves large areas devoid of epithelium, while sinuous furrows or fissures develop. These fissures then heal, the patches become pallid, and recovery takes place.

The various ulcerated surfaces so far described might be confused with ulcerative stomatitis, but their chronic character and insensitiveness as compared to acute ulcers of the tongue, associated with a specific history or manifestations of tuberculosis or syphilis elsewhere, render the diagnosis clear.

An ulcer on the frænum may be due to whooping-cough, in which disease the edge of the lower incisors may injure the tongue in the paroxysm of cough, or it may indicate the presence of a ragged tooth, which produces constant irritation, or, if the patient is advanced in years, represent the early stages of epithelioma, or that a broken pipe-stem has produced a wound.

Very rarely the tongue partakes of the ulceration of the tonsils and roof of the mouth which is seen in cases of Schönlein's disease, accompanied by purpuric eruptions on the skin and evidences of septicæmia.

Should the tongue be marked by bites from the teeth the patient may be an epileptic. Even if he denies that he is affected by the disease, the attacks may be unknown to him, because they are nocturnal. If the tongue is frequently bitten, the patient may be suffering from the early stages of glosso-labio-pharyngeal paralysis.[1]

The surface of the tongue may be attacked by various eruptions, such as measles, variola, eczema, herpes, erysipelas, pemphigus, zoster, or hydroa, and from the rupture of the vesicles or bullæ so formed ulcers may arise.

If the sore is herpetic, de Mussy asserts that the eruption will be found in the distribution of the lingual branch of the chorda tympani along the under border at the side.

[1] It may be pointed out in passing that if there be fits, and no biting of the tongue ever occurs, and the patient is a female, the attacks are probably hysterical.

Sometimes the surface of the tongue is here and there devoid of epithelium, and in some of these patches excoriated. Pain may or may not be present. The condition is called chronic superficial glossitis by Hack, and is considered by some to be the same disease described by Kaposi as glossodynia exfoliativa. It is more common in men and lasts many years.

Urticaria of the tongue has been reported by Laveran and xeroderma pigmentosum by Keating.

The presence of a plaque on the anterior portion of the dorsum of the tongue to one side of the median line, which is raised, not ulcerated, but red and irritated-looking, may be due to excessive smoking, the smoke irritating the local epithelium. It is always very smooth, later covered by a yellowish-brown coat, and is sometimes called "smokers' patch." It may extend over the whole tongue and last for years.

When the tongue has on its dorsum and edges dull-white or slate-colored dots, patches or lines, which are elevated, hard and horny to the touch, but not painful, the condition is known as leucokeratosis buccalis, or leucoma or ichthyosis, and this may arise from smoking or glass-blowing. It rarely begins in persons under twenty or in those over sixty years. It is often a strong predisposing agent toward cancer of the tongue. These spots are arranged on the tongue in longitudinal lines. Hyde asserts that they are due to excessive keratinization of the epithelium covered by an adherent and dense pellicle. The history is chronic, and ultimately by the stiffness of the spots the tongue may become cracked, and this in turn, perhaps, give rise to carcinoma. When the tongue is covered by smooth, dense plaques and disks or rings, the condition may be lichen planus, but the diagnosis of lichen planus from leucokeratosis buccalis is difficult, if not impossible. The plaques are most commonly seen in males between twenty and forty years. Closely allied to this is the rare condition of hardening of the tongue due to scleroderma, as described by Kaposi.

A very rare condition of the tongue is one in which its surface is marked by rings or areas on the dorsum, which gradually enlarge until they reach the edge or coalesce. In appearance they are red and smooth, deprived of filiform papillæ, but not of the fungiform variety. Often the border of the circle is more red than the centre, and the very edge is often yellowish. This condition is sometimes called wandering rash, geographical tongue, or annulus migrans.

Little if anything is known of its cause, save that delicate children are most often affected.

Feeble, sickly children sometimes develop upon the tongue, as well as on the lips and cheeks, a condition in which a tenacious exudation is thrown out, the mucous membrane becoming fissured and sore. Gaston and Sebestre have called this stomatitis impetiginosa.

Œdema of the tongue, with the development upon it of vesicles, and, finally, sloughs, may occur, and is probably identical with the foot-and-mouth disease of domestic animals.

Bilateral atrophy of the tongue is due to disease affecting the hypoglossal nerves in some part of their existence in or below the nuclei (see Paralysis of the Tongue). It occurs as a symptom of glosso-labio-pharyngeal paralysis, in which case the tongue is shrivelled and atrophied in patches, and in the later stages of the disease the organ has a crenated appearance. In other cases it is present in progressive muscular atrophy, and rarely in locomotor ataxia. It has also been seen in general paralysis of the insane. Unilateral atrophy may also occur from these causes, and Remak asserts that it sometimes arises from chronic lead-poisoning. Any disease involving the hypoglossal nerves may so result (see Paralysis of the Tongue).

In cases where the tongue is much enlarged the increase in size may be due to malignant growth, to macroglossia, which is a form of congenital lymphangioma, inflammatory hypertrophy, and syphilis, or acute inflammation from irritant poisons or foods. It may also be due to dermoid cyst, fibroma, lipoma, papilloma, angioma, myxoma, osteoma, and enchondroma. When it is due to acute glossitis the organ is seen to be several times its normal size, is protruded from the mouth, and marked by the pressure of the teeth. The organ is also clumsy and stiff, and heavily coated on the back portion. There is a profuse flow of saliva, and swallowing and speech are almost impossible. Glossitis may also be due to mercurialism, to septic infection, and may be either unilateral or bilateral. The tongue may be greatly enlarged by actinomycosis, this condition in olden times being called angina Ludovici. Great enlargement of the tongue may also arise in acromegaly and in myxœdema. In the latter disease the organ is broad, flat, and soft.

The movements of the tongue depend upon its innervation and its muscles, and afford valuable information in diagnosis. The rapidity of its protrusion in nervous and excitable persons when they are asked to show the tongue is noteworthy, and its constant rolling

is often seen in persons who are feeble-minded. In all diseases associated with mental hebetude its protrusion on request is very slow, although the patient will often do this act when all other orders fail to produce a response. In the various forms of coma due to apoplexy, diabetes, uræmia, and cerebral congestion, this condition obtains, and it is very characteristic of typhoid fever. Often the tongue which has been partially protruded is left so, even when the patient is told to draw it in. When the patient finds it difficult or impossible to remove food from between the teeth and cheek by means of his tongue, and complains that the power of speech is interfered with, because the tongue is clumsy in its movements, he may be suffering from the disease known as glosso-labio-pharnygeal paralysis or progressive bulbar paralysis. These lingual disorders are often the earliest signs of the disease. More rarely this disability of the tongue may arise from pseudo-bulbar paralysis, or what has been called glosso-labio-pharyngeal cerebral paralysis, a disease in which foci of softening occur in that portion of the cortico-muscular tract in which are the fibres which supply the muscles used in swallowing and speaking. This false type is separated from the true bulbar palsy by its sudden onset, an apoplectiform seizure, and other evidences of cortical disease. The tongue affords the most important points for differential diagnosis when a differential diagnosis is to be made under these circumstances, for in the false disease it does not waste or develop the reactions of degeneration, whereas in true bulbar paralysis these changes always speedily develop.

Paralysis of the Tongue. In apoplexy the tongue is protruded toward the paralyzed side as it is also in the condition already described of hemiatrophy. The lesions of the hypoglossus which produce paralysis may be of cortical origin (unilateral), in which case the hemorrhage or other injury may be situated where the middle and inferior frontal convolutions form the anterior central convolution,[1] or in the supra-nuclear tract between the cortex and the medulla, or in the hypoglossal nucleus, or, again, in the infra-nuclear tract within the medulla. Insular sclerosis may very rapidly cause lingual paralysis. Paralysis of the tongue may also result from injury to the hypoglossal fibres outside the medulla through meningitis or syphilitic or other growths. In still other cases pressure upon the nerve in its foramen may cause unilateral paralysis, or wounds of the neck, caries

[1] This is probably a fact, but not yet confirmed by autopsy, unless we consider Edinger's case of softening under this area, which affected the tongue only, as a typical one.

of the first cervical vertebræ, or cervical tumors may so result. Often in such a case the spinal accessory nerve is also involved. Very rarely, indeed, the tongue may be paralyzed by a hypoglossal neuritis (Erb). In rare instances hemiatrophy of the tongue is associated with hemiatrophy of the face without hypoglossal injury (Gowers). Girard asserts that the sensory part of the trifacial contains trophic filaments for the tongue, and that unilateral wasting may be due to disease of this nerve. In paralysis of the facial nerve the tongue may be partially paralyzed through the fact that the lingualis muscle is supplied by means of the chorda tympani nerve. When a tongue which is paralyzed unilaterally is retained in the mouth, it is seen that its root on the paralyzed side is higher than the other, owing to the paralysis of the posterior fibres of the hypoglossus, but when it is protruded the tongue goes toward the paralyzed side because it is pushed out by the fibres of the genio-glossus muscle on the well side. Finally, let us remember that if the tongue is paralyzed on one side the lesion is in the cortex or the pons on the opposite side of the body, or in the nucleus in the medulla on the same side of the body, or in the nerve after it has left the medulla. If it is bilateral paralysis the lesion is probably nuclear, because the nuclei are so closely situated that even a small lesion involves both of them, or it may be due to symmetrical disease of both sides of the cortex, the so-called pseudo-bulbar paralysis already spoken of.

It should not be forgotten that paralysis of the tongue may occur as the result of diphtheria.

Hirt asserts that the reaction of degeneration may be found in the tongue whether the lesion be cortical or in the nucleus. If the lesion is only cerebral this reaction will probably appear very late.

A tremor seen in the tongue may indicate a variety of nervous ailments or severe acute disease, as in typhoid and other severe infectious diseases, but the freedom from excessive coating and the absence of the ordinary signs of acute illness will separate the case of tongue tremor of acute disease from the tremor representing nervous ailments.

An important point to be regarded in noting lingual tremor is whether the tremor or fibrillary movement is constant, or whether it appears only when the tongue is moved to and fro or protruded. In typhoid fever the tremor occurs on movement, whereas in glosso-labio-pharyngeal paralysis when the mouth is opened fibrillary movements of the organ are often marked, while the organ lies in

the floor of the mouth powerless and beyond the control of the patient. Tremor of the tongue is also seen in a marked form in many cases of alcoholism, and associated with this tremor it will be noted that the protrusion of the organ is uncertain or in jerks.

Spasm of the tongue may be unilateral or bilateral, most commonly the latter. It is seen very commonly in cases of chorea, particularly of the post-hemiplegic type, and in hysteria. In the first disease the movements are characteristically choreic. In the latter the spasm may be tonic or clonic or alternately tetanic and irregular. Often the spasm in hysteria is unilateral. Sometimes it is clonic in puerperal melancholia. Spasm of the tongue is a common symptom in association with the twitching of the lips of general paralysis of the insane. Jerky movements of the tongue may also occur in insular sclerosis, but this is not the cause of the peculiar speech of that affection.

Very rarely the condition of lingual spasm is due to irritation of the hypoglossus by some cause as yet unknown. The tongue is darted in or out or thrown from side to side and often injured by the teeth. The spasms, as a rule, are not constant, but come on in attacks which closely resemble epilepsy, in that they are preceded by an aura (Remak and Berger). A very rare affection termed aphthongia (Fleury) is characterized by spasm of the tongue on attempting to speak. Romberg has recorded a case of lingual spasm due to irritation of the fifth nerve from lingual neuralgia.

In that very rare condition called "Thomsen's disease," "characterized by tonic spasms in the muscles during voluntary movements," the tongue may be involved, but in this case the other voluntary muscles will share in the affection.

Having considered the diagnostic significance of changes in the appearance of the tongue in this chapter, and of the appearance of the lips in the chapter on the Face and Head, there is yet to be discussed the condition of the buccal mucous membrane, the tonsils, the soft palate, the teeth, the upper part of the pharynx, and the post-nasal spaces. As almost all the conditions found in the latter regions are of interest to the rhinologist rather than the general practitioner, only one or two affections of these parts will be included in this work.

Swelling and redness of the buccal mucous membrane occur in the various mild forms of stomatitis, and in the ulcerative type of this disease the more severe lesions are often found in this area. In the malignant ulcerative stomatitis called noma the slough which

separates from the inside of the cheek leaves a large excavation which may become so deep as finally to perforate the cheek.

It is interesting to note that swelling of the cheek with great inflammation of the buccal mucous membrane is sometimes seen as the result of the formation of a salivary calculus in the duct of Steno, and it is also stated that obstruction from inflammation of this duct often occurs as a result of poisoning by sulphuric acid.

Again, in that rare disease called Schönlein's disease, or true peliosis rheumatica, the writer has seen a case, in which, in addition to the multiple arthritis, purpuric eruption, and great œdema, the formation of a large ulcer or slough threatened to perforate the cheek, and in healing produced a cicatrix which interfered with the patient's ability to open the mouth. This patient was an adult.

If a patient presents himself to the physician with the statement that he is suffering from general pains all over the body, particularly in the small of the back, quite high fever it may be, with much sore throat and difficulty in swallowing, the trouble in the majority of cases will be, in the adult, tonsillitis of the follicular form. If the symptoms are exceedingly severe, the inflammation may result in suppuration—suppurative tonsillitis. It is to be remembered in all cases that the systemic or constitutional disturbance is out of all proportion to the severity of the local lesions. If it is tonsillitis, the glands can be felt in the majority of cases a little beneath and forward of the angle of the jaw, and pressure upon them may produce considerable pain. If the mouth is well opened and the tongue depressed, there will be found on each side of the throat a large projecting and inflamed mass, in the depressions or follicular openings of which will be found a white or yellowish exudate, which in severe cases may spread over the surface of the gland till it slightly resembles the membrane of diphtheria. Pressure on the tonsil may cause the further projection of these cheesy-looking masses.

In the suppurative form of the disease the surface of the gland may be smooth and reddened, and in a day or two become soft and fluctuating, and if lanced pus will escape.

The severe constitutional disturbance, the soreness of the throat, difficulty in swallowing, and the follicular exudate call to mind in all such cases the possibility of the disease being diphtheria; but in tonsillitis the exudate can be easily removed without leaving a bleeding surface behind it, and it has not the dusky, dirty look of diphtheritic membrane. Again, in tonsillitis the exudate is seen on the

tonsils only, whereas in diphtheria it spreads over the half-arches and uvula. The general symptoms may make one suspect the onset of scarlet fever, particularly if the patient be a child; but the examination of the throat in scarlet fever shows the intense redness of the pharyngeal mucous membrane with comparatively slight enlargement of the tonsils. The lymphatic glands of the neck may be enlarged in scarlet fever, but are rarely so in tonsillitis.

If the patient first complains of dysphagia, and, on examination, the pharynx is red, and the tonsils are covered with patches which speedily spread, as just described, so that by forty-eight or seventy-two hours the tonsils, pillars, and soft palate are covered by a gray membrane, the case should always be diagnosed as diphtheria, and treated as such unless a bacteriological examination of the exudate shows the infection to be due to a streptococcus and not to the Klebs-Loeffler bacillus. Even if the patient has not true diphtheria he may be exceedingly ill. Again, it is to be remembered that while most of the cases of scarlet fever which present a membranous pharyngitis or tonsillitis are due to the streptococcus and not to the Loeffler bacillus, that in a certain proportion of these cases the two diseases, diphtheria and scarlet fever, exist simultaneously. Rarely a false membrane due to streptococcus infection, or still more rarely to the diphtheria bacillus, complicates the course of typhoid fever.

If in any case of diphtheria the false membrane extends to the nasal chambers, the prognosis is very unfavorable.

Ordinary sore throat or acute pharyngitis is generally accompanied with little systemic disturbance, the local pain and soreness being the most characteristic symptoms. Inspection will show the pharyngeal wall red and angry-looking and very likely unduly dry. Care should always be taken, in the case of children particularly, that the early sore throat of measles and scarlet fever is not taken for simple pharyngitis. Often the rash of measles can be seen on the pharyngeal wall some hours before the rash appears on the skin.

Sometimes cases are seen in which there are tonsillar pain and irritation, in which careful examination proves the discomfort to be due to the presence of a small calculus in a follicle of the tonsil.

When swelling of the tonsils is chronic the enlargement of these bodies may produce mouth-breathing, with the peculiar facies of that habit, deficient thoracic and general systemic development, and a peculiar cough, constant in character and worse at night. Often the

swollen or enlarged glands extending across the pharynx actually touch one another (see illustration in chapter on Face).

Finally, we can sometimes gain some information from the teeth as to the state of the patient. In children who are sufferers from rickets the teeth decay very early and rapidly, and if they be sufferers from inherited syphilis, the teeth are often cut in the early months of extrauterine life.

Caries of the teeth to an undue extent is also seen in many pregnant women and in cases of diabetes mellitus.

If the permanent upper incisors are notched or pegged-shaped with notches in the free edge, as if cut out with a small gouge, they are a fairly sure indication of syphilis of a hereditary character (Hutchinson teeth), and if in association with this deformity of the teeth we find middle-ear catarrh and keratitis, we have the "syphilitic triad," which is infallible as a sign of hereditary syphilis. These notches are not found in the so-called milk-teeth.

Fig. 57.

Hutchinson teeth.

The staining of teeth by tobacco or other materials held in the mouth may reveal certain habits of the patient, and a blue-line on the gums where they join the teeth will be an indication of the presence of chronic lead-poisoning. Loosening of the teeth with bleeding, spongy gums should call to the physician's mind the possibility of scurvy or scorbutus, and the spongy gums are particularly indicative of this affection in bottle-fed babies. If it occurs in adults, it may be due to mercurial salivation.

CHAPTER VI.

THE EYE.

The general diagnostic indications afforded by the eye—Diplopia and disorder of the external ocular muscles—Strabismus and squint—Disorder of the internal ocular muscles—The pupil—Hemianopsia—The visual fields—Color-vision—The optic nerve and its lesions—Retinitis—Amblyopia and blindness.

THE eye affords more information for diagnostic purposes concerning the condition of other organs of the body than any single part which can be examined. We gather from it not only a clear idea as to its own state, and the state of the nervous centres more or less intimately connected with it, but in addition we often gain positive information as to the condition of organs more remotely situated, as, for example, the kidneys. The parts of the eye which give us the greatest amount of knowledge about changes in other tissues are the optic nerve and retina, and the ocular muscles. The crystalline lens, the conjunctiva, and cornea often give additional evidence indicating the general systemic condition. Prominence of the eyeball or exophthalmos is seen as an almost constant symptom of true goitre, which for this reason is called exophthalmic goitre. Associated with the bulging eyeball we find more or less enlargement of the thyroid gland, an irritable heart, and a very rapid pulse, throbbing carotid arteries, marked general nervousness, often mental depression, and insomnia. In well-marked or advanced cases of exophthalmic goitre we often have a condition in which the upper eyelid does not follow the eyeball in its downward movement. This is sometimes called "Graefe's symptom." Again, there may be almost total absence of winking as an involuntary act, "Stellwag's symptom."

On examining the exterior of the eyeball we often notice a grayish ring along the junction of the cornea and sclera. It possesses when a complete ring but little significance, except age; but if it is but the segment of a ring or in two segments, one above and the other below the cornea, it is a true *arcus senilis*, and indicates in some cases fatty degeneration of the tissues of the body. The one is an annulus

senilis, the other an arcus senilis, and the arcus is the change worthy of note, although clinicians deny that either has any significance.

In addition to these objective symptoms we have also a very important set of signs connected with the ocular muscles, external and internal, as manifested by the various forms of strabismus or changes in the pupil and accommodation of the eye, by the ptosis already discussed in the chapter on the Face, and in nystagmus and ocular spasm. Beyond this, too, we have two other ocular symptoms subjective in nature, namely, diplopia or double vision and partial or complete blindness.

Diplopia depends upon the fact that in an eye in which the muscles are abnormal in their function the image which falls upon the fovea, or visual acuity spot of the retina, in the well eye fails to fall upon the same spot in the weak eye. To the well eye the object appears to be in the direction in which the eye is turned, whereas to the weak eye it appears to be in another direction. As a result, the mind gets the impression of two objects instead of one. The impression made on the well eye is the "true image" as it is called, and that in the diseased eye is called the "false image." Any cause which interferes with the fixation of each eye on the same point produces diplopia, and, as the eyes are normally directed to the object fixed by the ocular muscles, paralysis of any one of these muscles produces diplopia when the axis of one eye is deviated from the point of fixation, because the eye on one side is not properly moved by reason of the fact that one muscle has failed. Diplopia is ordinarily a constant sign of ocular muscle paralysis ; but if only weakness or insufficiency of a muscle is present, diplopia may never be a symptom recognized by the patient. The forms of diplopia—that is, the position of the false images in respect to the true images—vary with the muscles affected, and will be studied in a moment when paralysis of the muscles is tested for and their diagnosis discussed. It only remains at this place, therefore, to point out the probable significance if a patient with diplopia is presented to a physician.

Thus, a patient with diplopia may be suffering from a lesion in the cerebral cortex, such as hemorrhage, sclerosis, or softening ; or from a lesion in the cranial nerve nuclei, in the pons or corpora quadrigemina, or in the fascicular fibres. Again, diplopia may arise from lesions at the base of the brain, as meningitis, tubercular or syphilitic, or from injury to the nerves in the orbit or in their peripheral endings. As a result, we find diplopia in any disease which

may affect these parts, and it is quite a common symptom in locomotor ataxia, in Friedreich's ataxia, and in paretic dementia. Probably it is seen most commonly in ataxia, and with it, as the oculomotor nerve in its branch supplying the levator palpebræ is particularly apt to be paralyzed in this disease, we may find ptosis.

Diplopia is also found in cases of ptomaïne-poisoning, and in poisoning by belladonna, spigelia, conium, and gelsemium, owing to their effects on the ocular nerves.

The differential diagnosis between the various lesions producing diplopia is to be made by the other symptoms and the history of the case.

Paralysis of Ocular Muscles. As something has already been said in the chapter on the Face and Head of the diagnostic import of paralysis of the ocular muscles in connection with the subject of ptosis, a further consideration of the abnormal changes in their functions will be discussed first in the present chapter.[1] Before doing so, however, it is necessary to describe the methods resorted to for the purpose of demonstrating or determining departures from the normal in these muscles. In the first place, it must be clearly understood that the function of the extrinsic muscles of the eyeball is to direct the ball toward the object at which the patient desires to look, and they also evenly balance one another to keep the eye steady in its axis. Thus, the external and internal rectus muscles maintain the horizontal equilibrium of the eyeball. If the internal rectus is completely paralyzed in one eye, we have developed a unilateral external squint, the eye looking toward the outer side of the orbit; and if the external rectus fails, the eyeball is turned toward the nose. If these muscles are affected in both eyes, we have a divergent squint in the first case and a convergent squint in the second. Not only do the muscles of each eyeball govern the eye-movements of that side, but by the nervous centres governing the eye-muscles the two sets of eye-muscles are co-ordinated, so that they move as one organ in health.

Just here it is well for the reader to make a clear distinction between concomitant and paralytic squint, for they are two very different things in origin, symptoms, course, and prognosis. A concomitant squint is a wrong relation of the visual axes, so that they do not intersect in the point looked at; but there is no marked

[1] In the preparation of this chapter free use has been made of the article of my friend, Dr. de Schweinitz, on "Diseases of the Cranial Nerves," in Dercum's Nervous Diseases.

limitation of the movements of either eye in any direction. Be the direction of the eyes what it may, the squint remains practically unchanged. Further, if the fixing eye is covered, the other eye promptly fixes, and the covered eye deviates without the patient altering the position of the eye (Jackson). On the other hand, paralytic squint is the deviation which takes place when the attempt is made to turn the eyes in certain directions by means of the muscles which are paralyzed in whole or in part. When the attempt is made, the eye with the sound muscles turns as it should, while the eye with the paralyzed muscle hangs back, beginning to deviate as the eyes are turned, so that this muscle is required to perform its function, and deviates more as greater effort is required. The degree of squint and of separation of the double images it causes varies with the direction in which the eyes are turned, there being none at all in certain directions.

We examine the functional activity of the ocular muscles by the following measures :

The patient is told to look at the tip of a pencil or the tip of the finger of the physician, held about three feet from his face. This object is then gradually brought nearer and nearer to him, and the eyes of the patient necessarily converge more and more as it approaches the nose. Normally the eyes will be co-ordinately converged when the object is only three and a half inches from them; but if any weakness or insufficiency of one internus is present, the eye on that side will deviate or fail to converge before this point is reached.

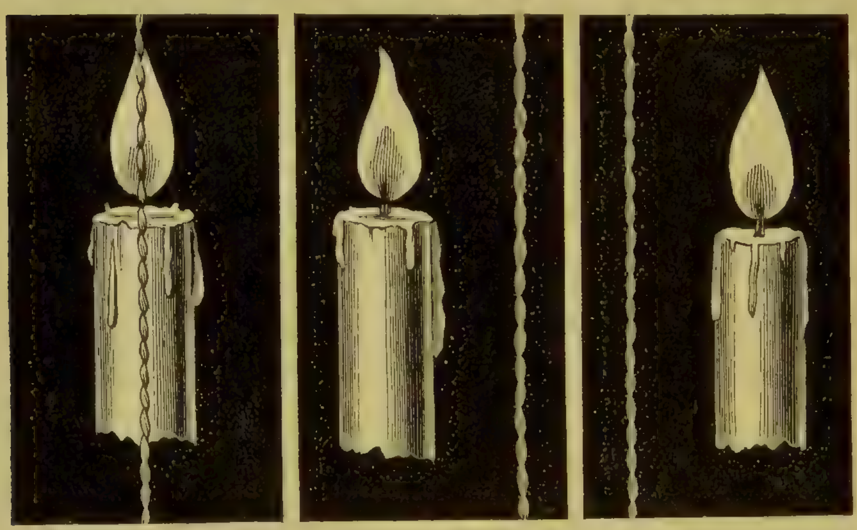
Again, a fine point like a pin-point is held about eight to ten inches from the eyes and below the horizontal, and one eye is covered by a card or the hand. If the eye which is separated from the object by the card deviates inward, it indicates insufficiency of the external rectus. If, on the other hand, it deviates outward, it shows insufficiency of the internal rectus. On sudden removal of the card the eye at once springs back into place for the purpose of fixing upon the object, and "in general terms each millimetre of movement deviating from the fixation-point corresponds to what is called two degrees of insufficiency, as measured by prisms" (Randall). If the internus is insufficient, and the covered eye moves in to fix in several distinct impulses, each impulse should be multiplied into the foregoing result.

A very useful, and the simplest, apparatus for testing the functional balance of the ocular muscles is the rod-test of Maddox.

A cell in which is mounted a transparent glass rod is placed in a trial frame, which is then placed in front of the eyes. If the horizontal deviation is to be determined, the physician must "seat the patient at six metres from a small flame, and place the rod horizontally before one eye, a colored glass before the other. If the line passes through the flame, there is orthophoria (equipoise), as far as the horizontal movements of the eyes are concerned. Should the line lie to either side of the flame, as in most people it will, there is either latent convergence or latent divergence : the former, if the line is the same side as the rod (homonymous diplopia) ; the latter, if to the other side (crossed diplopia)." (Maddox.) (Fig. 58.)

FIG. 58.

A B C



Maddox's rod-test for horizontal deviation. The rod is before the right eye. A. The line passes through the flame—orthophoria. B. The line passes to the right of the flame—latent convergence, or esophoria. C. The line passes to the left of the flame—latent divergence, or exophoria. (DE SCHWEINITZ.)

When the vertical deviation is to be estimated the rod is placed vertically in the flame. If the patient states that the horizontal line of light passes directly through the flame, the vertical balance of the eyes is normal ; if, on the other hand, the line is above the flame, there is a tendency to upward deviation of the naked eye ; but if the line is below the flame, there is upward deviation of the eye covered by the rod-test. (Fig. 59.)

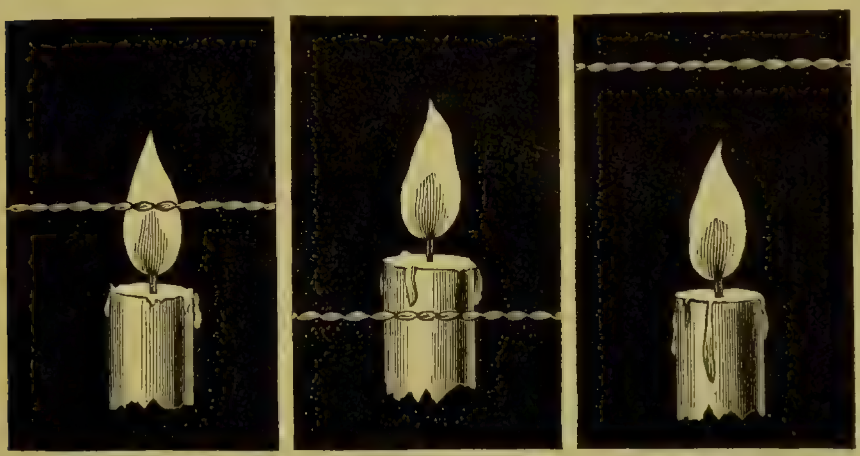
Testing of this kind refers to the insufficiencies and not to the palsies of the ocular muscles.

The importance of being able to demonstrate these minor failures

in the ocular muscles by these means lies in the fact that in this manner headaches due to muscle eye-strain may be remedied by removing their cause by properly fitted glasses, or by gymnastic exercises with prisms, or in some cases by tenotomy.

FIG. 59.

A B C



Maddox's rod-test for vertical deviation. The rod is before the right eye. A. The line passes through the flame—orthophoria. B. The line passes below the flame. The upper image belongs to the left eye—right hyperphoria. C. The line passes above the flame. The upper image belongs to the right eye—left hyperphoria. (DE SCHWEINITZ.)

Where there are marked *palsies* of the ocular muscles there is usually some poison exercising its effects upon their nervous centres or the nerves themselves, or there is some central nervous lesion affecting the centres governing these muscles in the cortex, or there is a lesion in the nuclei or fasciculi, or, again, there may be lesions in the basal ganglia, or in the course of the fibres of the nerve between the nucleus and the eye, or in the orbit or nerve-endings.

The signs of paralysis of the ocular muscles consist in the following symptoms: Diplopia, which is due to the failure of the images to fall on the corresponding points in each retina. This diplopia becomes more and more marked as the object is moved toward the side on which the paralyzed muscle lies. Strabismus, which may or may not be constant, usually develops when the patient endeavors to turn his eyes in the direction of the paralyzed muscle. Vertigo, which is due to the diplopia, or, if the well eye is closed, to an erroneous localization of the objects in the field of vision. Altered carriage of the head, due to the fact that the patient tries to turn his head in the direction in which he is least troubled by double

images; that is, he obtains the natural fixation-point of the weak eye, and then adjusts the well eye accordingly.

If the paralysis of the ocular muscle be complete, the squint and the loss of movement of the muscle which is paralyzed will usually enable the physician to find out the paralyzed muscle; but if there be only a partial paralysis or paresis of an ocular muscle, then squint is not necessarily present, and the diagnosis of the part affected must be made by a study of the double images. This is made by placing before the patient, at a distance of from three to five yards, a candle on the same level as his eyes. One eye is covered by a piece of red glass, so that the patient can readily distinguish between the images. The lighted candle is then moved from the middle of the patient to the right and left, and the relative positions of the red and white images noted. Then the candle is moved up and down, and the results recorded. These operations having been recorded, it is to be remembered that diplopia is most marked and sometimes only appears when the patient turns his eyes in that direction which calls into play the affected muscles, no

FIG. 60.

Paralysis of left abducens in a case of hemiplegia of syphilitic origin. (DERCUM.)

diplopia being present if other muscles are used. Again, the image which belongs to the affected eye is projected in the direction toward which the paralyzed muscle normally turns the eye, and, finally, the distance of the double image increases when the eyes are turned in the direction of the action of the paralyzed muscle, or, in other words, that image is false and belongs to the affected eye which in

the region of diplopia moves faster than the moving test-object—that is, the candle-flame.

If we place a candle several yards (say three to five) in front of a patient suffering from paralysis of the external rectus (Fig. 60) and at the level of his eyes, the double images of two candles will appear as in Fig. 61, if he has complete paralysis of the external rectus or

FIG. 61.

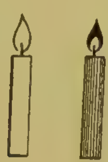
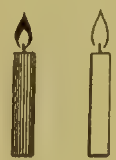


FIG. 62.



The false image is in outline.

internal squint of the left eye; while if the right external rectus is paralyzed, the images will appear as in Fig. 62. Further, if the object is moved to the right in the first condition, the false and true candle separate further and further; whereas if the left externus is involved and the object is moved to the left, the same separation takes place. This condition is called homonymous diplopia, because the word homonymous indicates that the false image is seen on the same side as the eye affected.

FIG. 63.

FIG. 64.

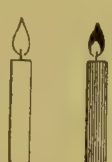


FIG. 65.

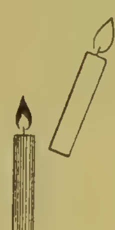


FIG. 66.

The false image is in outline.

If, on the other hand, the false image is found to the right of the true one, as in Fig. 63, the internal rectus of the left eye is paralyzed, and causes an external squint if the paralysis is complete; and if this same muscle of the right eye is affected, the false image will also be to the left side of the true one. (Fig. 64.) Further, if the object is moved to the right in the first case, the two images separate more and more widely; or if the right internus is involved, and the object is moved to the left, the same thing occurs. This is called crossed diplopia, because the image of the right eye appears on the left side, and the image of the left eye appears on the right side

Supposing, again, that the images are seen as in Fig. 65, then the left superior rectus is involved (downward squint); while if they appear as in Fig. 66, the right superior rectus is affected. This diplopia occurs chiefly in the upper field, because, according to a rule already given, diplopia is most manifest in that portion of the field of fixation toward which the paralyzed muscle commonly rotates the eye.

FIG. 67.

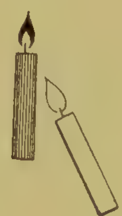


FIG. 68.

The false image is in outline.

If the images appear as in Fig. 67, the left inferior rectus is affected; or if as in Fig. 68, the right inferior rectus muscle. This is also crossed diplopia, chiefly in the lower field, because the inferior rectus muscle rotates the eyeball downward.

Again, if the images appear as in Fig. 69, the left inferior oblique muscle is paralyzed, and there will be inward and downward squint; if the right inferior oblique is affected, the images will be as in Fig. 70. There is a homonymous diplopia most marked in the upper field.

FIG. 69.

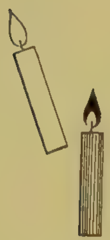


FIG. 70.

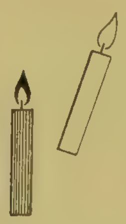


FIG. 71.

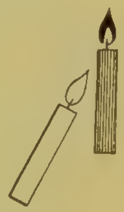


FIG. 72.

The false image is in outline.

If due to paralysis of the left superior oblique, the images appear as in Fig. 71; and if, as in Fig. 72, the right is affected. There is an upward and inward squint, and there is a homonymous diplopia chiefly in the lower field.

Finally, if there is divergent squint with failure of movement in all directions, except outward and slightly downward, and there are

ptosis, moderate mydriasis, and paralysis of accommodation, there are oculomotor paralysis and crossed diplopia.

The following table of Hotz (*International Clinic*, vol. iii., 4th series) summarizes the facts as to the diagnosis of the conditions producing strabismus :

I. Lateral diplopia indicates paralysis of an internal or external rectus.

1. Homonymous diplopia indicates paralysis of an external rectus.

a. Images separating to the right indicate paralysis of the externus of the right eye.

b. Images separating to the left indicate paralysis of the externus of the left eye.

2. Crossed images indicate paralysis of an internus.

a. Images separating to the right indicate paralysis of the internus of the left eye.

b. Images separating to the left indicate paralysis of the internus of the right eye.

II. Vertical diplopia in the upper field indicates paralysis of the superior rectus or inferior oblique.

1. Homonymous images indicate paralysis of the inferior oblique.

a. Image of right eye higher means paralysis of the inferior oblique of the right eye.

b. Image of right eye lower means paralysis of the inferior oblique of the left eye.

2. Crossed images indicate paralysis of the superior rectus.

a. Image of right eye means paralysis of the superior rectus of the right eye.

b. Image of right eye lower means paralysis of the superior rectus of the left eye.

III. Vertical diplopia in the lower field indicates paralysis of the inferior rectus or superior oblique.

1. Homonymous images indicate paralysis of the superior oblique.

a. Image of the right eye higher means paralysis of the superior oblique of the left eye.

2. Crossed images indicate paralysis of the inferior rectus.

a. Image of the right eye lower means paralysis of the inferior rectus of the right eye.

b. Image of the right eye higher means paralysis of the inferior rectus of the left eye.

It is exceedingly difficult, however, always to localize exactly the affected muscle, a difficulty which is much increased when more than one is paretic, the paresis being of different degrees.

Having now considered the means of determining that the muscles are defective, we must determine the diagnostic indications presented by this examination. In other words, we must seek the cause of the paralysis or loss of power.

Paralysis of the ocular muscles may be due to a lesion in one of several places. Thus it may arise in hemorrhage, sclerosis, and softening of the cerebral cortex, in which case the other symptoms of lesions in those parts will be present as in apoplexy, disseminated sclerosis, or meningeal disease. Or it may depend upon lesions in the fasciculi between the cortex and the nuclear origin of the nerves, as in the crus. (This is rare.) Or, again, it may be due to lesions in the nuclei. If this be the case, we have developed ophthalmoplegia,[1] or paralysis of all the ocular muscles supplied by the third, fourth, and sixth nerves. This nuclear paralysis is divisible into two classes, the acute and chronic. Sometimes it is called acute and chronic nuclear palsy. The acute form is sudden in its onset, all the ocular muscles losing power. With the onset of the attack there may be fever, vomiting, and even convulsions. Such an attack results from minute hemorrhage among the nuclei, or from an acute hemorrhagic polioencephalitis in the fourth ventricle, arising from syphilis, tuberculosis, ptomaïne-poisoning, alcoholic and sulphuric-acid-poisoning. Such cases are usually rapidly fatal. A less fatal form follows injuries, and the effects of nicotine, lead, carbonic acid, or such diseases as diabetes, syphilis, and epidemic influenza. Sometimes acute ophthalmoplegia comes on with acute poliomyelitis or acute bulbar paralysis.

Chronic nuclear paralysis is gradual in its onset, muscle after muscle failing, and even ptosis coming on. Sometimes after a certain degree of paralysis is reached the disease comes to a standstill. The trouble may be unilateral or bilateral, and is often unsymmetrical, and it results after acute ophthalmoplegia, as a congenital defect producing bilateral ptosis (see chapter on Face), as an acquired disease in childhood and adult life, and in conjunction with locomotor ataxia, paretic dementia, disseminated sclerosis, progressive muscular atro-

[1] Ophthalmoplegia is here applied in its strict sense. The word is often used to signify loss of power in individual eye-muscles; and while its use in both ways is correct, it is better to confine its usage to nuclear and complete lesions.

phy, chronic bulbar paralysis, and in connection with paralysis of the frontalis and orbicularis palpebrarum, which are innervated by the facial nerve. The cause may be tuberculosis or syphilis, but in some cases no cause can be found.

If the cause of the paralysis of one or two muscles be basilar lesions, these may arise from hemorrhage, pachymeningitis, meningitis, both simple and tubercular, chiefly the latter; purulent meningitis, abscess as the result of middle-ear disease, and anæmia. It may also arise as the result of obliterating arthritis, particularly in syphilitics, and from tumors. In children sudden convergent strabismus and diplopia are often among the earliest symptoms of tubercular meningitis at the base.

If the cause be in the nerve-trunks themselves, the lesion will probably be cellulitis, tenonitis, hemorrhages in the orbit, or fractures of the orbit; or, again, there may be disease of the frontal sinus. If the lesion is distinctly peripheral, it may be due to rheumatism (when the external rectus is commonly affected), neurasthenia, or it may arise from uric-acid diathesis and gout. Further, such lesions may be due to influenza, diabetes, diphtheria, lead and alcohol, or any one of the drugs which paralyze the ocular nerves.

So much for general statements as to the common and possible sites of the lesions producing paralysis of the ocular muscles. We can now go further than this and locate the lesion more accurately from the knowledge we have gained as to the particular muscle affected and the other symptoms presented by the case.

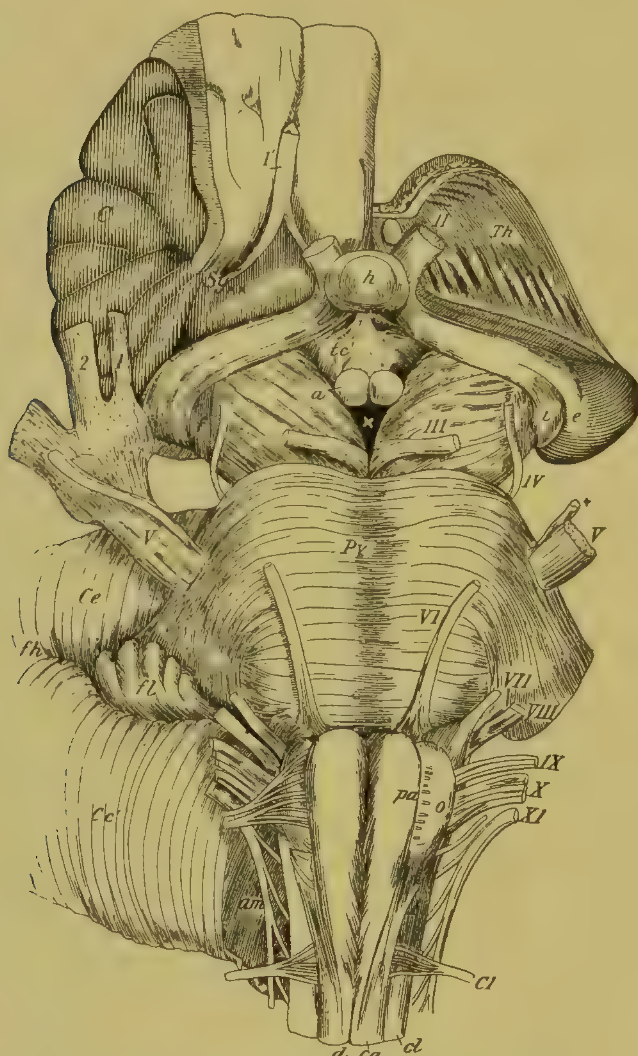
Let us suppose that a patient suffering from paralytic internal squint, or a diplopia which indicates paralysis of the external rectus, presents himself to the physician, what diagnostic significance has this symptom? [1]

In the first place, it is to be remembered that the external rectus receives its nerve-supply from the abducens, or sixth nerve, which arises from the pyramidal body close to the pons. (Fig. 73.) Its deep origin is a nucleus under the floor of the fourth ventricle. The nerve pierces the dura mater on the basilar surface of the sphenoid bone, passes through the clinoid process, enters the cavernous sinus, and, finally, enters the orbit through the sphenoidal fissure between the heads of the external rectus. If this form of squint is associated with hemiplegia of the opposite side of the body, the lesion

[1] This refers to paralytic and not to concomitant squint.

is in the pons on the same side as the affected eye and the opposite from the hemiplegia, because the eye-fibres have crossed higher up, but the motor tracts for the limbs cross lower down.

FIG. 73.



The base of the brain and the cranial nerves, crura, pons, and medulla. (ALLEN THOMPSON.) *I* to *XII*. The cranial nerves. *fh*. Optic thalamus. *h*. Pituitary body. *tc*. Tuber cinereum. *a*. Corpora albicantia. *P*. Pes pedunculi. *i*. Interior *e*. Exterior geniculate body. *Pr*. Pons Varolii. *pa*. Anterior pyramid of medulla. *o*. Olive. *d*. Decussation of anterior pyramid. *ca*. Anterior column of spinal cord. *cl*. Lateral column of spinal cord. *Ce*. Cerebellum. *fl*. Flocculus of cerebellum. *VI*. The sixth or abducens nerve.

On the other hand, if there is no monoplegia and abducens palsy (internal squint) on the same side of the body, the lesion is in their point of origin in the cortex, or, in other words, the lesion has taken foci above the point where the tracts cross. Such a paralysis is, therefore, cortical.

If, again, there is complete unilateral paralysis of the abducens (internal squint), with loss of the associated action of the internus, the lesion is in the nuclei under the floor of the fourth ventricle, because the nuclei of the third and sixth cranial nerves are closely connected, so that a lesion involving the sixth nucleus weakens the nucleus of the third nerve. (Fig. 74.) Complete paralysis of the externus may, therefore, be due to a nuclear lesion; for if the lesion were above the nucleus, this nucleus might obtain collateral impulses, as seen in this diagram, and, therefore, the paralysis would only be

FIG. 74.

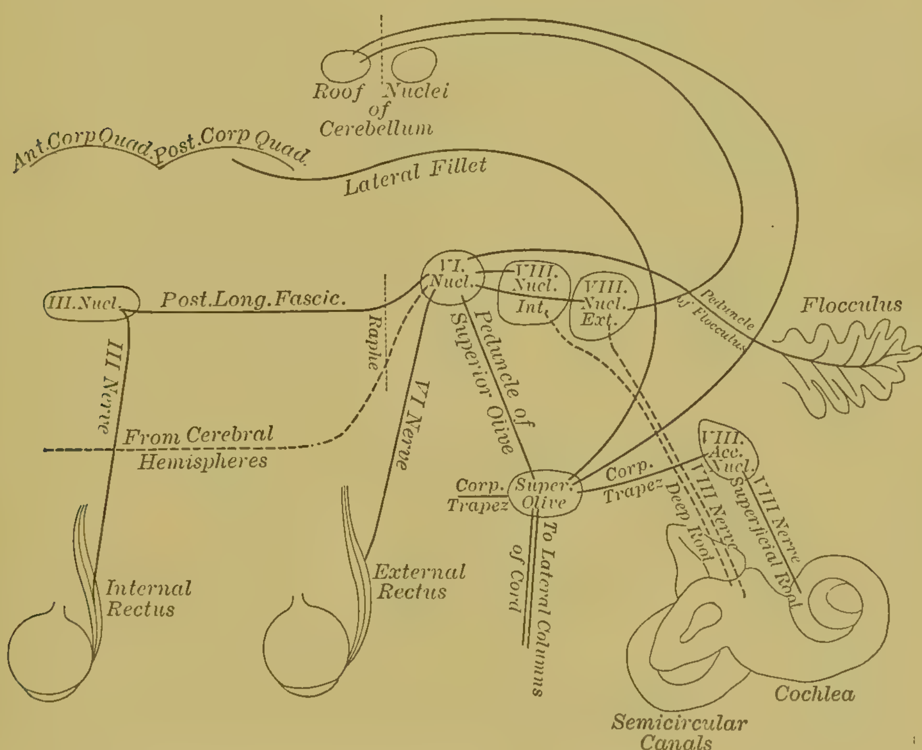


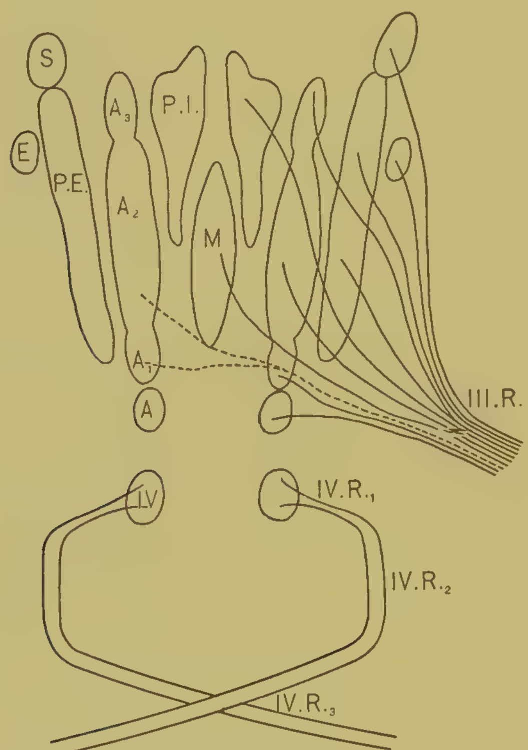
Diagram of the connections of the nucleus of the sixth nerve. (BRUCE.)

partial. It may also be due to a peripheral lesion. Sometimes, however, an inflammatory process pressing upon the basilar surface of the sphenoid, and thereby involving the nerve, may cause a similar effect. Loss of power of the external rectus may also arise from neurasthenia, uric-acid diathesis, gout and rheumatism, and in tubercular or syphilitic meningitis at the base, as already stated. It also comes on in some cases of diabetes, *la grippe*, and in chronic poisoning by lead and alcohol, or the acute poisoning of gelsemium, ptomaïne-poisoning, conium- and spigelia-poisoning.

Again, let us suppose that the internal rectus is paralyzed, causing

external squint. We remember that it is supplied by the oculomotor nerve, which arises from a nucleus in front of the corpora quadrigemina, which extends from the level of the posterior commissure to a point near the nucleus of the fourth nerve or patheticus. (Fig. 73.) Landois states that these two nuclei (the third and fourth) are united. The nucleus of the oculomotor nerve has been divided into several groups, as shown in Fig. 75, after Bruce, where, however, it is seen

FIG. 75.



Scheme of the segments of the nucleus of the third nerve and their relations to each other and to the nucleus of the fourth nerve. III. R. Third nerve. M. Median nucleus. A. Anterior nucleus, interior part. A_1. Anterior nucleus, lower part of main nucleus. A_2. Anterior nucleus, intermediate portion. A_3. Anterior nucleus, upper portion. P.I. Postero-internal nucleus. P.E. Postero-external nucleus. E. External nucleus. S. Superior nucleus. Some of the root-fibres from the lower and intermediate parts of the anterior nucleus are represented by dotted lines as crossing to the opposite side. IV. The nucleus of the fourth nerve. IV.R.1, IV.R.2, IV.R.3. The first, second, and third portions of the root respectively. (BRUCE.)

that the third and fourth nuclei are not united. The nerve itself pierces the dura mater below the posterior clinoid process, passes along the outer wall of the cavernous sinus, and after dividing into two branches enters the orbit through the sphenoidal fissure. The upper branch supplies the superior rectus and the levator palpebral, and the lower one after dividing into three branches supplies the internal rectus, the inferior rectus, and the inferior oblique muscles.

The oculomotor nerve receives filaments from the cavernous plexus of the sympathetic, and from the first division of the fifth nerve. In addition to divergent squint there is, as already pointed out in the last few pages, in oculomotor paralysis, as additional symptoms, ptosis, mydriasis, and paralysis of accommodation. The lesion producing unilateral ptosis may be found in the cerebral cortex on the opposite side from the affected eye in the angular gyrus just below the interparietal fissure. Again, tubercular or other degenerative disease of the corpora quadrigemina may cause double ptosis.

If the patient has ptosis with preservation of the function of the intraocular muscles (that is, partial oculomotor paralysis), with hemiplegia of the opposite side of the body, the lesion, according to Mauthner, probably affects the fascicular fibres—that is, those between the cortex and the nuclei. There may be associated with this form of oculomotor paralysis loss of power in the hypoglossal and facial nerves. On the other hand, if the oculomotor paralysis is complete, the lesion is almost certainly at the base of the brain, and this diagnosis becomes practically certain if there is associated with it paralysis of other cranial nerves. Paralysis of the oculomotor nerve on one side with hemiplegia on the opposite side of the body is not positively a crus or fascicular lesion unless the paralysis occurs simultaneously. (Hughlings Jackson.)

If, however, there be double oculomotor paralysis, the lesion is bilateral and probably due to a lesion at the base, as meningitis or arteritis; or to an inflammatory exudate involving both nuclei; or, again, to diphtheritic poison or the lesions of tabes dorsalis.

If that very rare form of ocular muscle paralysis, namely, isolated palsy of the fourth trochlear nerve, is present, we will probably find that the paralysis is due to a lesion at the base of the brain, due to meningitis, or the pressure of a growth.

Supposing, however, that a patient presents himself with swelling of the eyelids, exophthalmos, a contracted, followed by a dilated, pupil, anæsthesia of the skin of the upper eyelid and of the temple, or the area supplied by the first division (ophthalmic) of the fifth nerve, and ophthalmoplegia—that is, paralysis of the extrinsic ocular muscles on one side—where will be the lesion productive of this train of interesting symptoms? It will be seen at once that such a condition is the result of paralysis of the oculomotor (third), pathetic (fourth), and abducens (sixth) nerves, and that as in all probability only one lesion has produced these symptoms it must exist at some point

where all these nerve-fibres are so closely approximated that they are readily involved together. It will be recalled that the course of these nerves is as follows: The oculomotor nerve having arisen from the nucleus in the corpora quadrigemina pierces the dura mater below the posterior clinoid process, passes along the outer wall of the cavernous sinus and there divides into two branches. The pathetic nerve passes near the clinoid process along the outer wall of the cavernous sinus and with the oculomotor nerve enters the orbit through the sphenoidal fissure. The sixth nerve pierces the dura mater on the basilar surface of the sphenoid bone, passes through the clinoid process, and enters the cavernous sinus, finally reaching the orbit through the sphenoidal fissure. It is thus seen that a lesion at the sphenoid fissure and pressure in the cavernous sinus would cause all the symptoms described above. This occurs in cases of thrombosis of the cavernous sinus. Where there is an arterio-venous aneurism of this sinus there will be pulsating exophthalmos. Injury or inflammation may also produce a series of symptoms if in this area.

The significance of conjugate lateral paralysis producing a deviation of both eyes to the right or left, as the case may be, is that some lesion exists in the cerebral cortex, the corona radiata, or the internal capsule, or in the pons before the fibres have crossed. The lesion, if in the cortex, however, does not have to be localized in one spot, for any source of irritation in the cortex may apparently cause conjugate deviation. If the lesion is the result of an apoplexy, the eyes are turned toward the side opposite to the paralysis (Prevost's symptom)—that is, the "patient looks at his lesion." The reason that a unilateral lesion can cause a bilateral deviation is that the lateral movements of the eye are governed by an impulse which passes down from the cortex to the sixth-nerve nucleus and thence across the posterior longitudinal fasciculus to the opposite side, where it passes to the nucleus of the third nerve. As conjugate lateral deviation is caused by contraction of the internal rectus on one side (third nerve) and the external rectus on the other (sixth nerve), the mechanism of the deviation is clear. Thus if the lesion be a distinctive one on the left side of the brain, causing right hemiplegia, the eyes will be turned to the left by the action of the unaffected left external rectus and the right internal rectus; while if the lesion be on the right side of the brain, the reverse will occur. If, however, the lesion be irritative (as a tumor), this deviation is reversed, because in this case

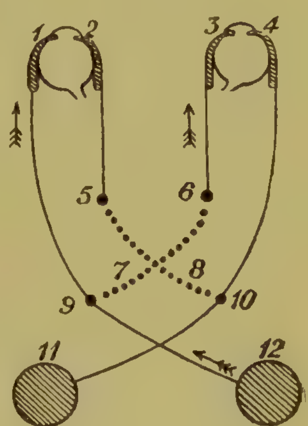
the centres are irritated and cause spasm of the muscles receiving their nerve-supply from the affected side of the cerebrum. In other words, the eyes are turned toward the side of the body which is convulsed.

In the first instance the eyes are turned away from the affected side because the muscles of the eyes on that side are also paralyzed, and the eyes are therefore turned by the muscles which remain intact. In the second instance the eyes are turned toward the convulsed side because the internal and external rectus are spasmodically contracted and so overcome the healthy muscles.

We find, however, that, if the lesion be in the pons rather than in the cortex, these conditions are reversed, for now a destructive lesion causes the eyes to be turned to the paralyzed side, and an irritative lesion directs them away from the paralyzed side.

This is best explained by the following diagram and description from Swanzy's well-known book. (Fig. 76.)

FIG. 76.



1. Left ext. rectus; 2. Left int. rectus; 3. Right int. rectus; 4. Right ext. rectus; 5. Nucleus left third nerve; 6. Nucleus right third nerve; 7 and 8. Post. longitudinal bands from sixth nerve to opposite third nerve; 9. Nucleus left sixth nerve; 10. Nucleus right sixth nerve; 11 and 12. Left and right cortical centres. An impulse starting from 12 would travel down to 9, and produce an associated movement of the eyes to the left.

A destructive lesion at 12, the right cortical centre, involving also motor centres of the body, would cause left hemiplegia; and, since the external rectus of the left eye and internal rectus of the right eye would be paralyzed, the antagonists would turn the eyes to the right; *i.e.*, away from the paralyzed side. A destructive lesion of the right side of the pons, also producing left hemiplegia, if it involves the sixth nucleus, will produce paralysis of the external rectus of the right eye and of the internal rectus of the left eye, and then the antagonists would turn the eyes to the left; *i.e.*, toward the paralyzed side. It is easy to see how irritative lesions would produce exactly the opposite effects.

Squint which is due to hysteria is always caused by spasmodic contraction of the eye-muscle and is never due to paralysis, as it

often is in organic disease. Very often there is a spasm of the eyelid or eyebrow associated with it. The administration of a relaxant, such as chloroform, will at once overcome such a squint.

Nystagmus, or the rapid oscillation of the eyes from side to side or in a vertical or rotary movement, is usually bilateral.[1] When not congenital it is a frequent symptom of disseminated sclerosis, Friedreich's ataxia, and advanced locomotor ataxia, and while it does not localize the lesion it indicates very positively that one is present and that the case is not one of hysteria or functional disease. Nystagmus occurring in children is very often associated with imperfect vision of great degree or with blindness as a result of opacity of the cornea, congenital cataract, or atrophy of the nerve. In other instances, however, it arises from growths in the cerebellum or pons, and it is sometimes seen in hydrocephalus and very rarely in acute meningitis and in epilepsy. Very rarely lateral nystagmus is seen in children who seem otherwise normal, and it then possesses no particular diagnostic importance.

Paralysis or Disorder of the Intraocular Muscles. Having discussed the diagnostic indications of changes in the function of the extraocular muscles, we next proceed to a consideration of these facts in connection with the intraocular muscles. These consist, it will be remembered, in the muscular fibres of the iris, circular and radiating, and the ciliary muscle. The nerve-supply of the iris consists in fibres from the oculomotor or third nerve, the upper or ophthalmic division of the fifth, and the sympathetic. It will be remembered that in the posterior part of the orbit there is situated a ganglion called the ciliary or ophthalmic ganglion. By its short or motor root this ganglion is connected with the third nerve, by its sympathetic root with the cavernous sympathetic plexus and the cervical sympathetic plexus, while by its long or sensory root it is connected with the nasal branch of the ophthalmic or upper branch of the fifth nerve. From this ganglion extend forward two sets of nerves, one short (the short ciliary nerve), which supplies the iris and the ciliary muscle, and one set long (long ciliary nerves), which also go to the iris. The filaments which go to the ganglion by means of its short or motor root (from the oculomotor nerve) pass forward to the circular fibres of the iris, while those which have arisen in the sympathetic plexus pass forward to the radiating fibres. These last

[1] The physician should remember that some occupations, such as mining, produce in some persons nystagmus without the presence of the disease about to be named.

fibres are in part derived from the cervical sympathetic ganglion, run through the carotid plexus, and are controlled to some extent by the cilio-spinal centre of Bunge in the spinal cord at about the seventh cervical or first dorsal vertebra.

The ciliary muscle is supplied by the fibres of the short ciliary nerves, which have arisen in the floor of the third ventricle and which is connected with the nucleus of the third nerve.

The size of the normal pupil is about 4 millimetres in diameter, but this varies according to the degree of light to which the patient is exposed. It ought always to be measured by a millimetre measure which gives its approximate diameter.

The pupil to be tested must be free from any abnormal conditions produced by new or old inflammation of the iris, and the light used should not be excessively bright, but about that usual to the eye.

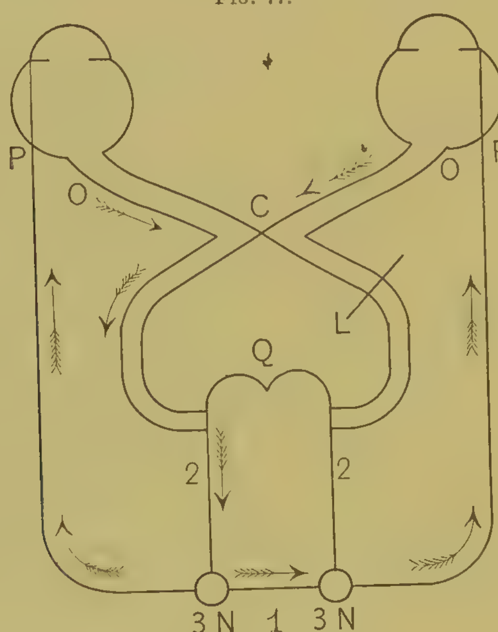
Testing the Pupil. The patient is to be placed facing the light and told to look at some distant object. The hands of the physician are then placed one over each eye, the patient being told to keep his eyes open and to endeavor to see the object seen before the eyes were covered. One hand is now quickly removed from one eye and the pupil observed. This observation must be acute or the pupillary contraction will occur before it is seen. This reflex is due to the fact that we have an irritation of the optic nerve by light, and this sends a reflex wave to the centres governing the pupil and causes it to contract. Not only does the uncovered pupil react in this manner, but the covered one does the same thing. The first is called a direct reflex, the second is called the indirect or consensual reflex. The exact pathway of this reflex is unknown, but we know that the light-impulse passes along the optic nerve, and, arriving at its semi-decussation in the chiasm, passes along each of the tracts to the corpora quadrigemina, and thence by the communicating fibres (Meynert's fibres) between these bodies and the oculomotor centres to the centre for the sphincter pupillæ or circular muscle-centre, and from there to the ciliary ganglion, the ciliary nerves, and the muscles of the iris. (Fig. 77.)

Not only does the pupil change its size by reason of the ordinary light-reflex, but it also contracts or dilates in association with the other muscles governing accommodation and convergence, namely, the ciliary muscle and internal recti. This is the associated reaction of the pupils and is tested by causing the patient to direct his eyes to a near object—for example, the point of a pencil. If the sight is intact, contraction of the pupil will occur.

The pupil-dilating centre is in the medulla and is very sensitive to reflex irritation.

Supposing that the pupillary movement is abnormal, we should recollect before studying the case further what the causes of its perversion may be. Thus its size is altered by drugs, by local disease of the iris, by spinal disease and disease of the sympathetic, by localized cerebral lesions, by abeyance of the cerebral functions, and by irritation of the brain. Let us suppose, however, that on test-

FIG. 77.



3N, centre of third nerve ; 1, connection between nuclei of third nerves ; 2, Meynert's fibres ; Q, corpora quadrigemina ; C, chiasma ; O, optic nerve ; P, myotic fibres of third nerve ; L, seat of lesion ; arrows show path of impulse in lesion of right tract at L. (SWANZY.)

ing the ocular reflexes in the manner already described, we find that the pupil of one eye when uncovered does not contract, but immediately does so as soon as the other eye is uncovered, what is the indication ? It indicates that there is disease of the optic nerve of that eye which does not convey the impulse of light from the retina ; whereas if it contracts when the other eye is uncovered, it shows that the rest of the mechanism involved in the reflex is intact. Accommodative reaction of the pupil is intact also. Again, supposing that irides fail to react to light, but do to accommodation and convergence, we have the "Argyll-Robertson pupil," so-called, which indicates that a lesion exists in the communicating fibres (Meynert's fibres) which convey the impulses from the corpora quadrigemina to the oculomotor nuclei. (See Fig. 77.)

This condition is seen in locomotor ataxia, general paralysis of the insane, sometimes in cerebral syphilis and as the result of poisoning by the bisulphide of carbon. It is worthy of note, however, that late in all these affections the reaction to accommodation may also be lost. Rarely the reverse of the Argyll-Robertson pupil occurs as the result of a lesion in the second and third parts of the oculomotor nucleus. If the eyes fail to react to light and to accommodation, there is probably blindness due to optic-nerve disease.

If on throwing light into the right eye there is no reaction of the pupil of that eye, and on throwing it into the left eye there is still no reaction in the pupil of the right eye, there must be a lesion of the nucleus of the right oculomotor nerve or palsy of the conducting fibres of each optic nerve.

Dilatation of the pupil occurs in cases of glaucoma, optic-nerve atrophy, in disease of the orbit, and under the effect of drugs possessing a mydriatic action, as, for example, atropine. It also occurs in persons suffering from fright, neurasthenia, aortic regurgitation, and irritation of the cervical sympathetic, as by aneurism. A dilated pupil is also often seen in idiotic children.

Dilatation of the pupil results from two causes, opposite in character: the first is irritation due to tumor, meningitis, or other irritating lesion of the upper part of the spinal cord; the second to paralysis of the cerebral centre of the oculomotor nerve, resulting from cerebral hemorrhage, thrombosis, tumors, or abscess of the brain.

Contraction of the pupil is also due to lesion similarly situated and results from sources of irritation in the cerebrum, resulting from meningitis and cerebral tumor, and Berthold asserts that a contracted pupil shows that a sudden attack of paralysis is due to embolism and a dilated pupil shows hemorrhage. Myosis (contraction of the pupil) results from paralyzing lesions of the spinal cord situated in the region of the cervical vertebræ, and occurs notably in locomotor ataxia. It is also seen in general paralysis of the insane (paretic dementia), the false paretic dementia of syphilis, and in bulbar paralysis with progressive muscular atrophy. It is also one of the most notable signs of opium-poisoning.

Under the name of "hemiopic pupillary inaction" or "Wernicke's pupil" we sometimes, though rarely, meet with a condition associated with hemianopsia or blindness in one-half of the eye, which is demonstrated in the following manner: The patient is seated in a dark room and one eye is covered. The other eye is now illuminated by

just sufficient light from a flat mirror to enable the physician to see the eye. By means of the concave mirror of an ophthalmoscope the physician now directs into the uncovered eye a bright beam of light, taking care that it falls upon one side of the retina, or, in other words, enters the eyes obliquely and strikes on the side of the retina which is blind. If when the light falls on the blind side of the retina there is no pupillary reaction, it is considered that the lesion exists in the right arc between the optic chiasm and the corpora quadrigemina; but if there is a pupillary reaction, the lesion must be further back in the visual centres, back of the reflex arc. When the lesion is found back of the reflex arc it may indicate a lesion of the optic tract, the posterior segment of the thalamus, the posterior part of the chiasm, or rarely it may be caused by a lesion of the optic nerve if the hemianopsia be monocular, which is rarely the case.

Finally a rhythmical contraction and dilatation of the pupil, called "*hippus*," are seen in health for a moment on sudden exposure to light; but when constant is a sign of disseminated sclerosis, epilepsy, or the early stages of acute meningitis. It is sometimes seen in hysteria.

The presence of a recurrent, unequal dilatation of the pupils of a transitory character is said to be by Rampoldi an early and almost constant sign of pulmonary tuberculosis. He claims that this is due to a reflex irritation of the nerves governing the pupil through the sympathetic system. Probably in these cases enlarged glands in the chest are the cause of the pupillary phenomenon, just as an aneurism may be. Destree claims that 97 per cent. of his cases of phthisis present this pupillary symptom.

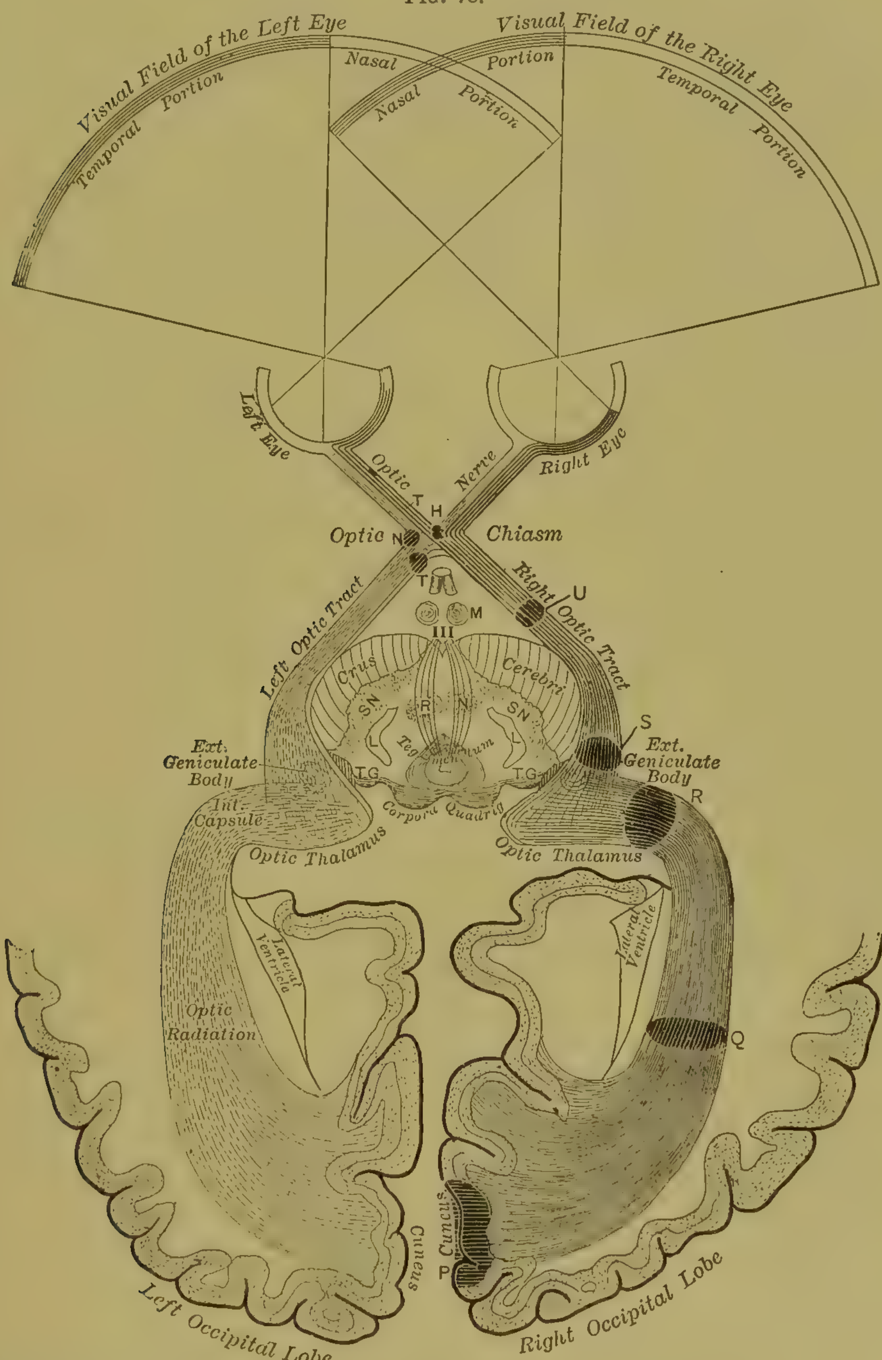
Knies points out that pupillary contraction and dilatation take place in association with Cheyne-Stokes breathing. Dilatation usually exists with the inspiratory movements, and myosis occurs during the interval of apnœa.

Changes in the Acuity of Vision. Having discussed the diagnostic value of alterations from the normal in the function of the extra- and intraocular muscles of the eye, we can proceed to a consideration of the value in changes in the acuity of vision. The questions of the acuity of vision in relation to errors in the refractive media of the eye will not, of course, be included in this book.

Failure of vision in part or *in toto* depends upon a lesion which destroys the peripheral ocular sense-organ (the eye), the optic nerves, the optic tracts, or the receptive and perceptive centres of sight. It

also is dependent upon bilateral lesions in the crystalline lens, as in cataract, or in the cornea, as in severe keratitis.

FIG. 78.



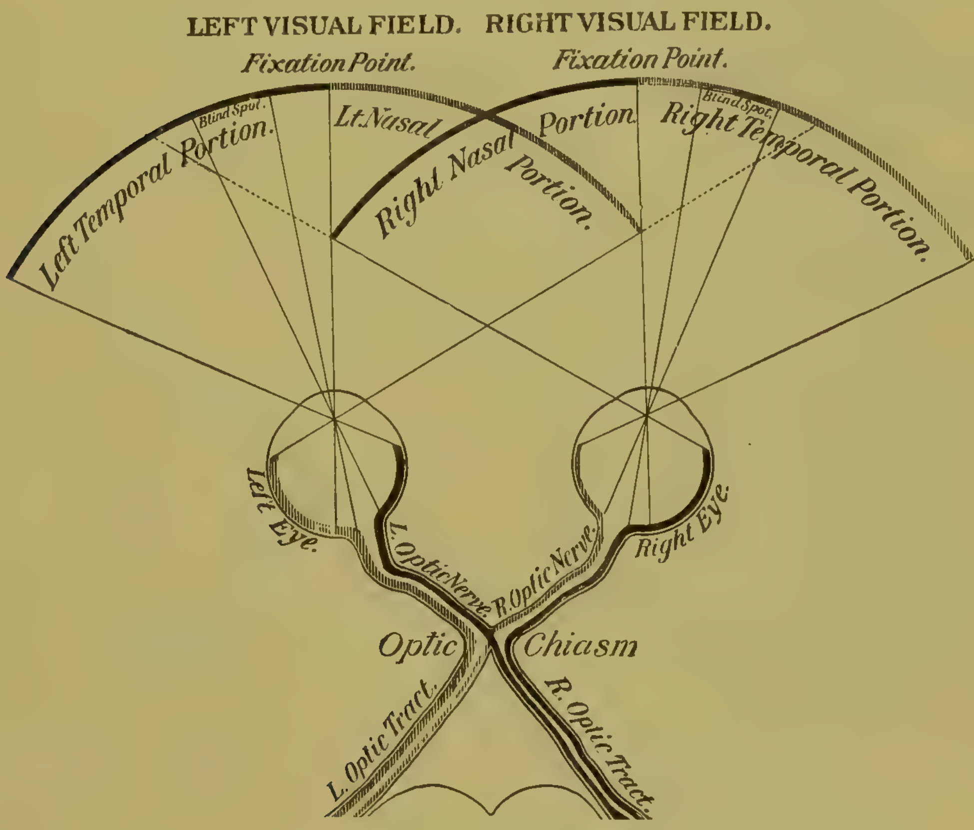
The visual tract. The result of a lesion anywhere between the chiasm and the cuneus is to produce homonymous hemianopsia. H. Lesion at chiasm causing bilateral temporal hemianopsia. N. Lesion at chiasm causing unilateral nasal hemianopsia. T. Lesion at chiasm causing unilateral temporal hemianopsia. SN. Substantial nigra of crus. L. Lemniscus in crus. RN. Red nucleus. III. Third nerves. P, Q, R, S, U. Lesions in the occipital lobe and in front of it producing left homonymous lateral hemianopsia.

Before we discuss these various causes of blindness it is necessary that we recall the nervous anatomy of the organs of sight. These nerve-fibres starting with the rods and cones of the retina and the fibres from the macula pass back along the optic nerve until they come to what is known as the chiasm, where the various fibres from the eye decussate, in that the fibres from the inner half of each eye cross to the opposite side, whereas those of the outer half of each eye pass to the same side, as is shown in Fig. 78. After the optic tracts have been formed by this (partial) decussation each one winds around the corresponding crus cerebri, and terminates in two roots upon the corpora geniculata externa and interna and upon the posterior part of the optic thalamus. The pupillary fibres also branch here to the corpora quadrigemina. These parts are known as the primary optic centres. After leaving them the fibres pass backward into the posterior part of the posterior limb of the internal capsule and thence to the cortex, rise in a fan-shape, pass outside the tip of the lateral ventricle, and reach the secondary or true optical centre in the lower part of the median aspect of the occipital lobe. (See Fig. 78.)

HEMIANOPSIA. As lesions of the nervous centres frequently produce partial or complete blindness, it is of importance, first, that the presence of partial blindness should be discovered, and, second, that the lesion causing it should be located. Aside from general failure of vision due to changes in the retina or optic nerve we have in many cases of nervous disease a condition called hemianopsia or partial or complete blindness of one-half of the retina. Usually hemianopsia is bilateral—that is, in both eyes; and it is usually homonymous—that is, on the same side of each eye; or, in other words, if it is the outer half of the left eye, it will be in the inner half of the right eye. If this is the case, it is called left bilateral homonymous hemianopsia. If, on the other hand, the outer half of each eye is blind, this is called bitemporal hemianopsia; or if the blindness is found in the nasal side of both eyes, it is called binasal hemianopsia. It must be remembered, however, that the apparent blindness of the outer side of the eye is really due to disease of the fibres supplying the opposite side of the retina, as is shown in Fig. 79. The presence of hemianopsia in any form is determined by the following method of examination: The patient is placed with the back to the light and one eye is covered, while the other is fixed upon the centre of the physician's face, which should be two feet away. The finger of the physician is now

moved to the left and right as far as the patient can see it, the head and the eyeball of the patient remaining fixed. If the eye fails to see the finger when but a little distance to one side or the other of the fixation-point, hemianopsia is present.

FIG. 79.



We measure the field of vision more accurately by means of what is known as a perimeter, which is a semicircular metal band which revolves upon its middle point, being capable therefore of describing a hemisphere in space. This arc is divided into degrees marked on it from 0° to 90°, and at the centre of it is placed the eye which is to be examined, which eye finds its fixation-point in the centre of the semicircle. A small piece of white paper is now moved along the metal arc on its inner surface, from the extremity and toward the centre, until it comes into view, when the physician notes the number of degrees at which the object is seen and notes it on a chart (see Fig. 80). The area of the normal field is well seen in this figure.

Let us suppose that on using the tests just described we find left

lateral homonymous hemianopsia—that is, blindness in the visual field, as shown in Fig. 81. This signifies that the patient has a lesion somewhere in the visual tract back of the chiasm, either

FIG. 80.

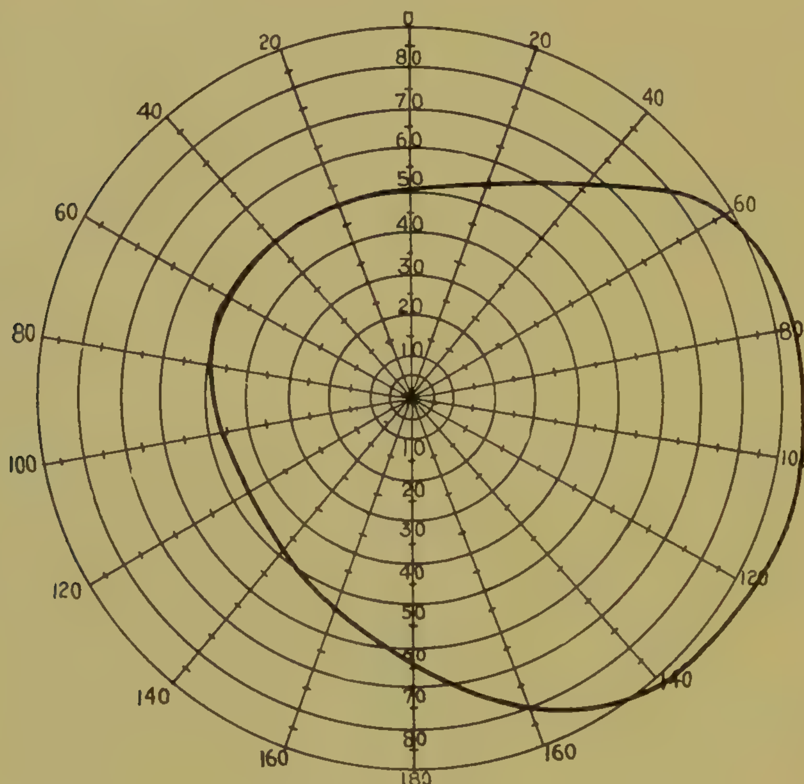
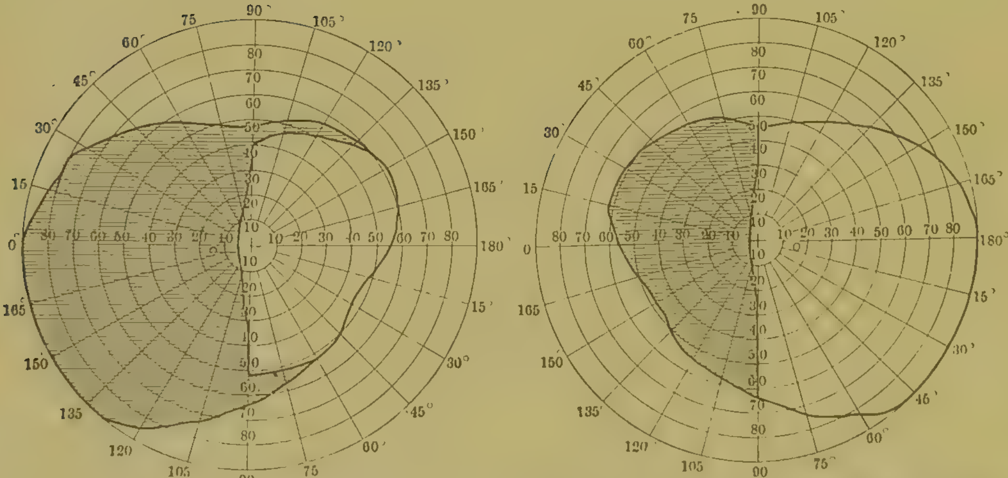


Chart of F. V. of right eye.

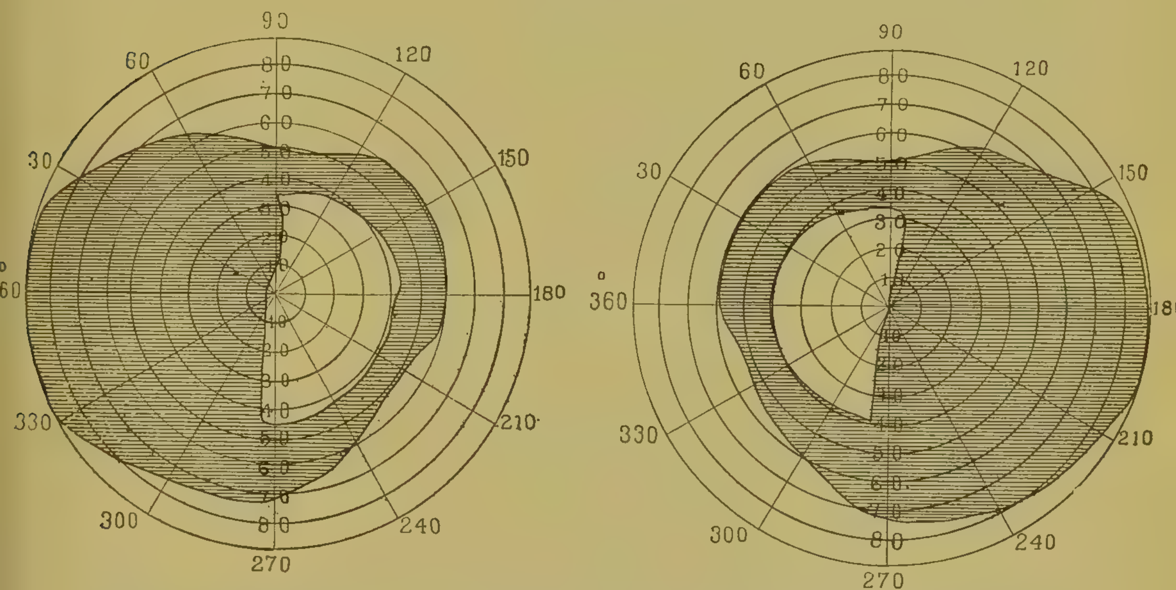
FIG. 81.



Left homonymous hemianopsia from a case of gunshot-wound, with suspected lesion of the right cuneus, from a case under the care of S. WEIR MITCHELL, at the Infirmary for Nervous Diseases. (DE SCHWEINITZ.)

in the cuneus, in the occipital lobe, in the optic radiations, in the internal capsule, in the primary optic centres, or in the optic tract. Fig. 78 shows the sites of these lesions and why they cause left homonymous hemianopsia. Supposing, on the other hand, that in place of left homonymous hemianopsia we find bitemporal hemianopsia (Fig. 82), this indicates that the patient has a lesion of the optic tracts in the crossing fibres in the middle of the chiasm (see "H" in Fig. 78); or if binasal hemianopsia, that he has a lesion on both sides of the chiasm or one on the outer side of each optic nerve. This is a very rare lesion.

FIG. 82.



Bitemporal hemianopsia from a case of akromegaly originally under the care of DR. H. C. WOOD and later studied by DR. PACKARD. Eyes examined in 1885 by DR. G. E. DE SCHWEINITZ. and above fields found. (DE SCHWEINITZ.)

Hemianopsia of the homonymous form is very rarely found in hysteria, generally in association with hysterical hemianæsthesia, in which condition the conjunctiva is usually anæsthetic, thereby differing from the condition of the conjunctiva of persons suffering from hemianæsthesia of an organic origin.

In some cases in place of hemianopsia we have simply an alteration in the visual fields for color. It will be remembered that the boundaries of the power of the clear perception of colors are not identical with the boundary for white light nor they are identical with one another. Passing from the periphery toward the centre of the visual field in ordinary daylight we find that blue is the color first seen, its boundary being almost as great as that of white. After

blue comes yellow, orange, red, and finally green. The blue, red, and green being the most important colors, their boundaries are shown in Fig. 83. These fields are determined by means of small pieces of colored paper passed around the perimeter in the manner already described.

FIG. 83.

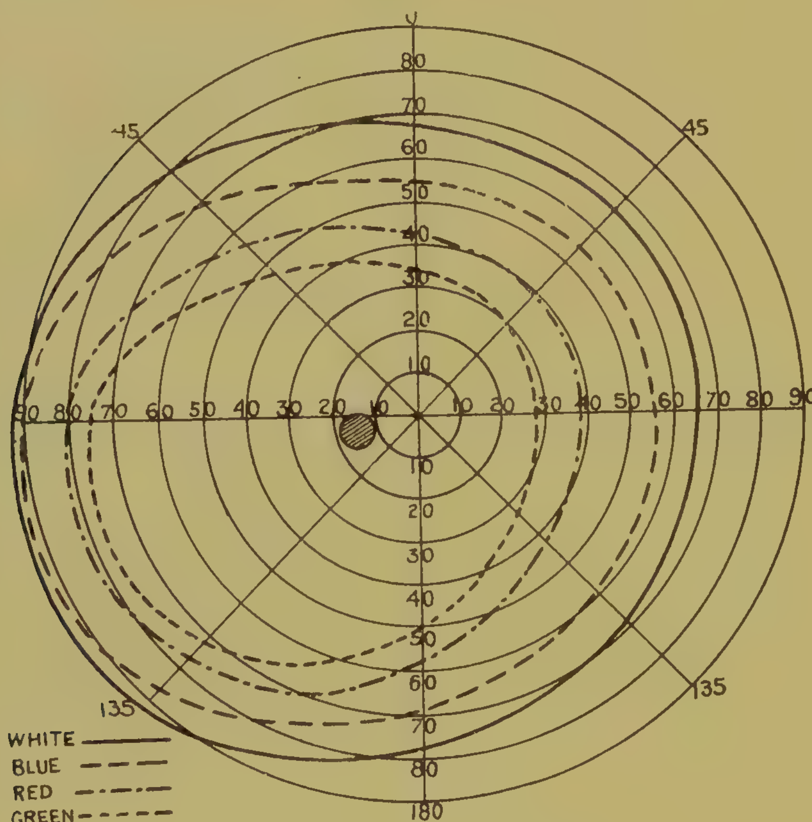


Chart of F. V. of left eye. (LANDOLT.)

The alteration of the visual field for colors is called, if so situated, homonymous hemidyschromatopsia, and the lesions producing it is situated in the cortex of the occipital lobe; while if the colors are indistinguishable, it is called hemiachromatopsia. This, however, has recently been denied. The transposition of the visual fields for color is usually a symptom of hysteria, and as a rule the red field takes the place of the blue, and *vice versa.* The fields for all the colors are also markedly narrowed in hysteria. This transposition, rather than loss of color-sense, helps us sometimes to a distinction between the ocular symptoms of hysteria and of true tabes dorsalis, a distinction which is of great importance, yet one which is often exceedingly difficult, save for these and two other symptoms, namely, that in hysteria the knee-jerks are usually preserved and the Argyll-Rob-

ertson pupil is not seen. The following table from Charcot's lectures for 1888–89 summarizes these differential points:

	Tabes.	Hysteria.
Motor apparatus of the eye.	Paralysis from lesion of a motor nerve of the eye (bulbar or peripheral; consequent diplopia.	1. Sometimes associated paralysis. 2. Blepharospasm. 3. Monocular diplopia; micropsia and macropsia.
Pupillary disturbances.	Argyll-Robertson pupil.	
Optic disk.	Atrophy.	
Symptoms due to affections of the optic nerve or visual centres.	1. Irregular concentric contraction of the visual fields. 2. *Tabetic* achromatopsia or dyschromatopsia, affecting first green and red, yellow and blue being preserved to the last. 3. Progressive blindness.	1. Regular concentric contraction of the visual fields. 2. Dyschromatopsia from simple contraction of the visual fields for colors. Frequently perception of red alone persists. 3. Transitory amblyopia or amaurosis.

The Optic Nerve and the Ophthalmoscope. There still remain to be considered the diagnostic indications afforded us by the optic nerve. Before taking up this subject mention must be made of the manner of using the ophthalmoscope.

The patient is to be seated in a darkened room, and by his side, at the level of the eye to be examined and far enough back of him for his face to be in shadow, should be placed a lamp, or, if gas can be had, an Argand burner. The physician now seats himself, if the right eye is to be observed, at the right side of his patient, and takes a chair slightly higher than that of the patient. The ophthalmoscope is now taken in the right hand and held in such a position that the concavity of the physician's brow fits over the convexity of the instrument. The eye of the physician is so placed that he can readily see through the aperture in the centre of the ophthalmoscope, and by means of the concave mirror on the face of the instrument he reflects the light into the eye through the pupil. The patient must not look directly into the ophthalmoscope, but to one side, and his vision should be distant and accommodation as far as possible relaxed. If the examiner is not skilled in the use of the ophthalmoscope and the result of the examination is of great importance in the diagnosis of the case, it is justifiable to use homatropine to dilate the pupil and prevent the alterations of accommodation by paralyzing this function. The ophthalmoscope and the head of the physician are now approached as closely as possible to the eye of the patient, the

angle of the two heads being as nearly as possible identical as shown in Fig. 84. If the light be now directed slightly toward the nasal side of the eye, the optic nerve will be seen, or in its stead a retinal bloodvessel will be seen across the field of vision, and this should be traced along its course to its origin in the papilla. If the patient or the physician is short-sighted (myopic), the ophthalmoscope must

FIG. 84.

Relative position of physician and patient whilst employing the direct method. (NORRIS and OLIVER.)

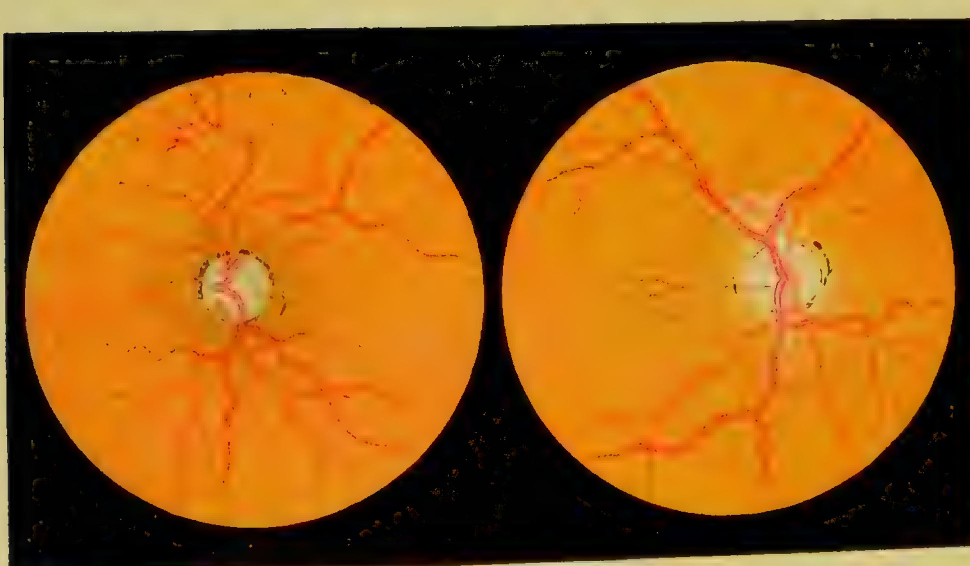
be adjusted to correct this error by placing over the aperture a concave lens; but if ordinary degrees of far-sightedness (hypermetropia) are present, the use of a convex lens is not necessary, because the accommodation of the eye makes up for the error in refraction. If the hypermetropia is so great that accommodation cannot overcome it, then a convex lens must be used. The view of the eye which is obtained ordinarily by a beginner is clouded, not because of myopia or hypermetropia, but because the physician has not as yet learned to relax his accommodation in making the examination. A concave glass usually remedies this.

PLATE IV.

FIG. 1.

Normal Eye-Ground (average tint). (Norris & Oliver.)

FIG. 2.



Primary Atrophy of the Optic Nerve (Spinal Atrophy). Modified from Haab (de Schweinitz.)

Post-Papillitic or Consecutive Atrophy of the Optic Nerve. Modified from Jaeger. (de Schweinitz.)

In health the optic nerve appears as a nearly round or slightly oval disk, situated somewhat to the nasal side of the eye, and varying in color from grayish-pink to red, the centre being whiter and the nasal half the darkest part. Around the papilla are seen two rings, the outer one darker and generally incomplete or absent, while the inner one is a faint white stripe, which becomes more marked as the patient grows older. The first is called the choroidal ring, and represents the edge of the choroidal coat of the eye where it is pierced by the nerve. The second is the scleral ring, which is the edge of the sclerotic coat. The centre of the optic papilla may be even with the surface or cupped and may be stippled or dotted in appearance. The retinal arteries emerge from this central spot and the chief venous trunks empty into it. Generally one arterial and one venous stream pass up and a similar one downward, and both soon bifurcate, afterward still further dividing. The arteries are distinguished from the veins by their bright-red hue, while the veins are darker in color. The veins are about one-third larger than the arteries. A bright stripe due to an optical delusion seems to divide each vessel longitudinally into two parts. The arteries of the normal eye do not pulsate, but pulsation of the veins is quite common. It must be remembered that the appearance of the papilla and of the bloodvessels as they leave it varies very greatly within perfectly physiological limits. As already stated, the cupping of the papilla may be quite deep or quite shallow, and the bloodvessels may divide as already described, or divide in the papilla into four branches. The veins are usually more tortuous than the arteries. (Plate IV., Fig. 1.) The retina is practically transparent so that the underlying choroid is seen. In persons with a dark skin the retina has a grayish hue in the neighborhood of the papilla, which is most marked on its nasal side and is slightly streaked.

To the outer side of the papilla, slightly below the horizontal meridian, is the macula lutea or yellow spot, which is about the size of the end of the optic nerve, but darker in color, somewhat granular, and devoid of any retinal vessel. It is the point of the eye-ground in which direct vision is best developed. In its centre is a bright spot, the fovea centralis. As a person grows older these clear distinctions vanish and the macula lutea is to be distinguished from the surrounding eye-ground only by its darker hue and the absence of vessels. The macula is difficult to see, because as the light falls on it the pupil at once contracts. If the eye is dilated by a mydriatic, how-

ever, and the patient looks directly into the ophthalmoscope, the macula is readily seen.

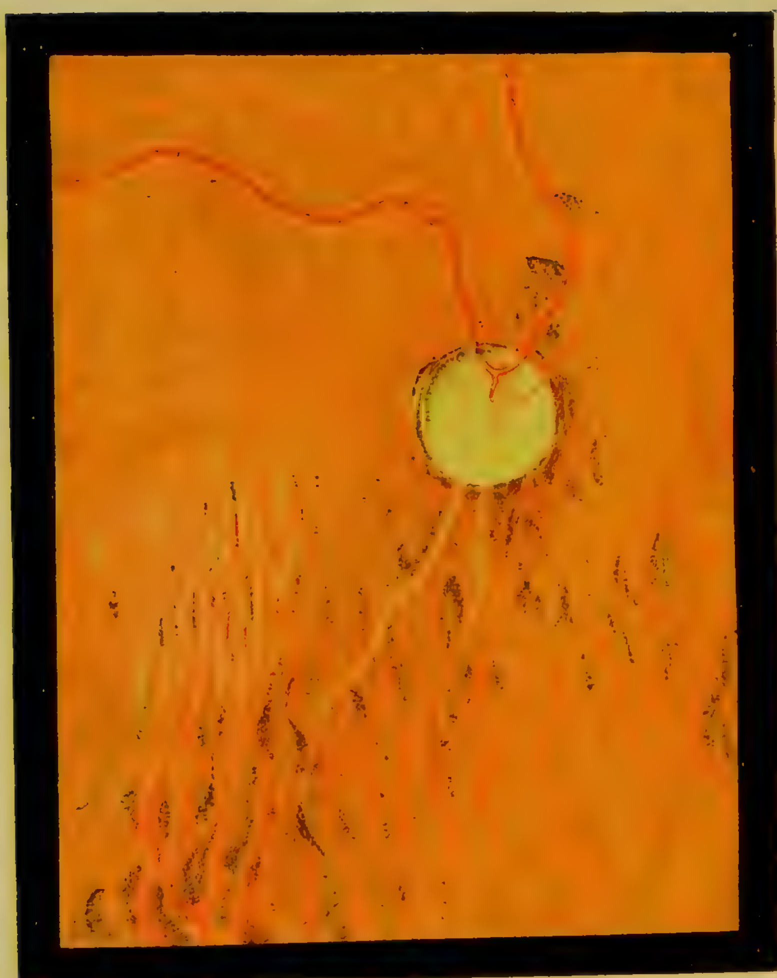
The red glare produced by throwing the light into the eye by the ophthalmoscope is due to reflection from the bloodvessels of the choroid coat.

The pathological significance of alterations in these normal appearances is very great. Let us suppose that on examining the eye-ground we find the end of the optic nerve red and its edges irregular and obscure, or if the morbid condition is further advanced and the nerve-head look protruding or mound-like and the arteries going to it are smaller than normal and partly concealed, while the veins are enlarged and tortuous. Hemorrhages may be seen in the papillary region or near it occurring in flame-like shapes. These are the signs of optic neuritis, and optic neuritis depends upon intra-orbital or intracranial disease, although if the process is not marked it may be due to hypermetropic astigmatism. Vision is often unaffected, but if the lesion be in the cerebellum sudden blindness may come on.

As some differences of opinion exist as to the various forms of neuritis of the optic nerve the term papillitis is often used to signify all the forms of optic neuritis which we meet with, or in other cases is spoken of as choked disk. Papillitis is more commonly the result of brain-tumor than of any other intracranial lesion, and, again, it is much more common in lesions of the cerebellum than in tumors elsewhere in the brain. Another fairly common cause of papillitis is meningeal inflammation, particularly about the base of the brain, and tubercular meningitis is very prone to produce it. Cerebral abscess may also cause this change in the optic nerve.

In addition to the cranial causes of papillitis we have acute febrile disorders, syphilis, toxæmias from lead and alcohol, rheumatism and anæmia. Sometimes, however, they produce an acute or chronic retro-bulbar neuritis. There is nearly always in such cases a large central scotoma, which causes a failure to recognize color, as, for example, green or red. Sometimes the patient realizes the failure of his vision which may be impaired otherwise than by disorder of the color-sense. In other cases he fails to do so until his eyes are examined. The chronic form of retrobulbar neuritis is generally the result of the excessive use of tobacco and alcohol, and produces what is called tobacco-amblyopia or toxic amblyopia, with failure of vision from these causes. In such cases there is a central scotoma

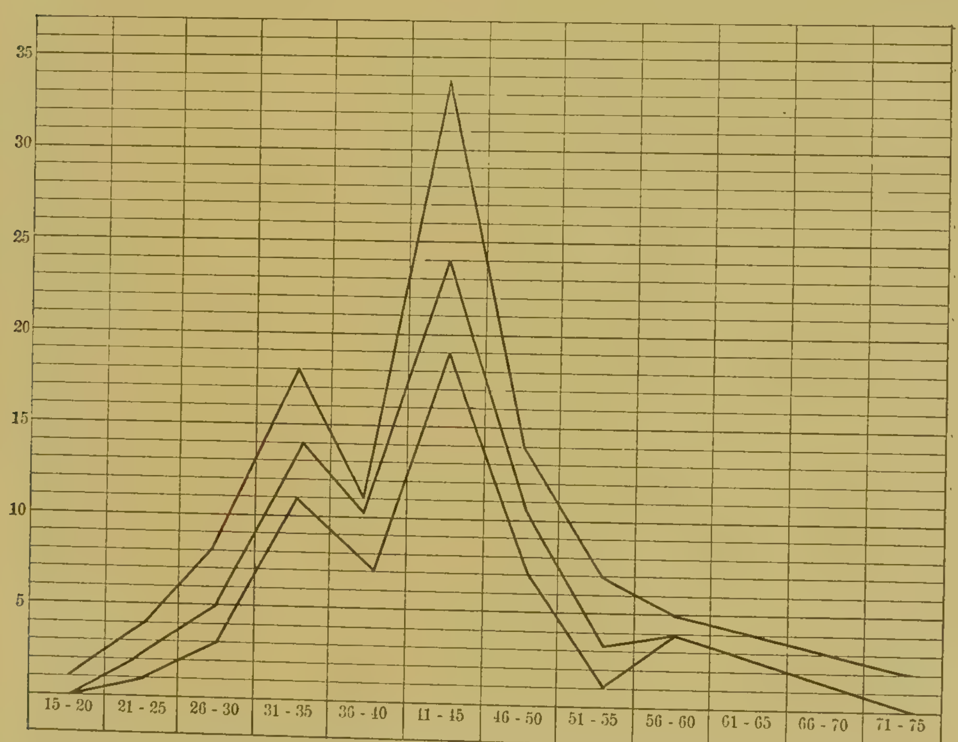
PLATE V.



Embolic Atrophy of the Optic Nerve. From a case in the Jefferson Medical College Hospital.
(de Schweinitz.)

between the macula and the optic nerve where the sense of red and green is lost. The ophthalmoscope may reveal in such cases discoloration of the disk and a triangular spot of atrophy in the outer and lower part of it. Supposing, however, on using the ophthalmoscope we find in place of a papillitis an atrophied state of the nerve, in which, if the disease be young, the nerve-ending looks gray and the outline of the disk is sharp (Plate V., Fig. 2), or if it be well advanced the edges appear hazy, the arteries contracted, and the veins large and tortuous, while the disk is quite white. (Plate V.) The primary or gray form of atrophy described is most typically seen in the optic-nerve lesion of locomtor ataxia, and so is often called tabetic atrophy. About 34 per cent. of all tabetics

FIG. 85.



Upper curve, frequency of tabes. Middle curve, frequency of severe ocular symptoms. Lower curve, frequency of atrophy of the optic nerve. (BERGER.)

suffer from this change. Again, it is seen in cases of paretic dementia somewhat less frequently. Optic atrophy is often seen in cases of disseminated sclerosis. Because of the fact that gray atrophy of the nerve is one of the earliest signs of locomotor ataxia, in some cases it is a valuable one in the diagnosis of this grave disorder, sepa-

rating it from pseudo-tabes due to ordinary peripheral neuritis. The following table, also taken from de Schweinitz's article on this subject, shows the relation between age, severe ocular symptoms, and atrophy of the optic nerve. (Fig. 85.)

The more advanced forms of optic atrophy with a hazy outline of the disk usually result from diseases in the optic centres or in the nerve itself. Thus there may be present a tumor pressing on the chiasm or optic tracts.

Again, if on the use of the ophthalmoscope we find that there is a faint haziness of the retina, that whitish streaks are found in it which may be bluish-gray or yellowish in hue, that the bloodvessels are tortuous and minute vessels are easily seen because of their enlargement, that hemorrhagic exudations of a flame-like character are present, and that dark pigmented spots represent where previous hemorrhages have been, and, finally, that the head of the optic nerve is not clearly outlined, we have the picture of retinitis. Generally, in association with these signs, we find as subjective symptoms changes in the visual field, a distorted vision, so that straight lines appear bent inward or outward, and there are pain and fear of light. If the vitreous humor is opaque, in addition to these symptoms, syphilis may be present, and the iris may give evidence of iritis. Where the hemorrhages are very manifest and profuse (hemorrhagic retinitis) the cause may be disease of the heart and bloodvessels.

By far the most important of these forms of retinitis from a diagnostic standpoint is what is known as albuminuric retinitis or that due to Bright's disease. Here, in addition to the flame-like hemorrhagic areas, we find irregular spatterings of white which may be star-shaped. The importance of the discovery of such changes is that by it is the first suspicion of renal trouble discovered. This is of the greatest value in pregnancy. Retinitis also sometimes results from diabetes.

Hemorrhages into the retina without retinitis usually are the result of septicæmia, ulcerative endocarditis, hæmophilia, diabetes, gout, and malarial fever of a severe type. It is also seen in cases of great cardiac hypertrophy with stenosis, and after suffocation.

The iris indicates disease in other organs more rarely than the retina and optic nerve and the muscles, but an irregular pupil indicating an old iritis should raise a question as to a history of injury, syphilis, or rheumatism.

Finally, it should not be forgotten that cataract sometimes occurs

as the result of diabetes mellitus and that corneal ulceration is often an evidence of scrofulous tendencies, while a distorted pupil due to an old iritis should raise a suspicion of syphilis.

It must not be forgotten that patients often have, in distinction from distorted images, visions or flames of light or bright sparks before the eyes, or in their place dark dots called muscæ volitantes. Often the visions are the prodromes of an attack of migraine or of an epileptic seizure. In the case of spots of light or stars we usually find them as a result of severe indigestion, and the dark spots may arise from the same causes. Muscæ volitantes may also be due to small particles of mucus floating over the cornea or to small floating bodies in the vitreous.

Partial or complete blindness is sometimes seen in cases which are under the influence of a drug, as, for example, quinine or other drugs; and sometimes partial or complete blindness results from uræmia (uræmic amaurosis). As a rule, it does not occur as a single symptom, but follows an attack of acute uræmic manifestations; that is, it is found after a convulsion or period of coma has passed by. As a rule, nothing abnormal is found in the eye to account for it, and the pupillary reflexes are intact. The effect of the poison in the blood is therefore exercised upon the optical centres, probably in the occipital lobe. Sight is usually regained in these cases in a few days.

CHAPTER VII.

THE SKIN.

The color of the skin—Eruptions on the skin—Gangrene, ulcers, and sloughs—Scars, sweating, dryness, œdema, hardness—Anæsthesia, and hemianæsthesia—Paræsthesia, hyperæsthesia, itching.

MUCH information can be obtained by careful examination of the skin in many cases of disease. The examiner should make a note of the color of the integument, of its general nutrition, of its pliability and its sensibility. Naturally the eye at once takes in any eruption or scars which may mar its naturally smooth surface, and, as eruptions and scars are often the manifestations of more or less active systemic disorders, an insight into the presence of internal disease may be obtained from them.

The color of the skin in health in the white race depends upon the presence of pigment in the cells of the mucous layer of the epidermis, and in the corium in those parts of the body where pigmentation is marked, or to the condition of the subcutaneous circulation or of the blood in the subcutaneous vessels. Thus we often find the skin of the perineum, scrotum, axillæ, and of the lower abdomen much darker than elsewhere in persons in perfect health. Similarly we see a marked reddish or yellowish-brown hue in those parts of the skin which have been exposed to sun and weather, as a result of a deposition of pigment and an increased capillary circulation. With these normal alterations in color, however, we have little to do, for it is the abnormal colorations which interest us from a diagnostic standpoint. The most common of these changes in color due to pigment is jaundice; the next the chloasma of pregnancy or uterine disease, a condition usually limited to the face. Abdominal growths due to tuberculosis, cancer or lymphoma, and tuberculosis of the peritoneum also cause pigmentation of the skin, and in melanotic cancer there is often very dark discoloration, so marked as to be confused with that of Addison's disease. Again, it is not uncommon for persons who have hepatic torpor with constipation to develop what are called liver-spots, in which the skin has rather

a dirty hue. Under the name of vagabond's "pigmentation," we sometimes see discoloration induced by the irritation of the skin produced by lice and exposure to dirt and weather, and this is capable of being mistaken for the pigmentation of Addison's disease. Finally, we see the yellowish-brown hue of the skin due to tinea versicolor, the bronzing of the skin in Addison's disease, and the slate-blue hue of argyria or chronic silver-poisoning. (See further on in this chapter.) The changes in color depending upon disturbance of the subcutaneous circulation or on alterations in the blood are either local or general. In extreme nervousness, flushing or blushing, due to a local vasomotor relaxation with increased blood-supply, may redden the face and neck, or in hectic fever a hyperæmia of the skin over the malar bones may give rise to an increase in color, which may be dusky red, due to imperfect oxidation of the blood. Considerable cyanosis of the face and hands in a case of tuberculosis of the lungs is a very grave symptom. Again, we see in pneumonia a peculiar dusky red flushing of one cheek or of the entire face, and in erysipelas the zone of hyperæmic redness is characterized by its sharp line of demarcation and its raised edge. In the alterations in color due to changes in the quality of the blood we have, as causes, anæmia due to lack of corpuscles or of hæmoglobin, arising from the various etiological factors producing such states.

Jaundice. Taking up the color-changes due to pigment, we find that in jaundice the deposition of the biliary coloring-matter varies in degree from a slight tinge or almost imperceptible yellowing to a dark citron or olive-green hue.

In examining the skin for jaundice care should be taken not to do so by gas or candle-light, for the yellow flame masks the biliary color; and if the tinge is very slight, it may be made more marked by stretching the skin on the palm of the hand or by pressing upon the skin a glass slide so that the yellow hue shows through it.

Having discovered that biliary coloring-matter has been deposited in the rete mucosum, it remains for the physician to decide what the cause of the jaundice may be. In the first place, it must be remembered that jaundice may be hepatogenous—that is, arise from disorder in the liver, or be hematogenous from disorders of the blood with the setting free of blood-pigment. The hepatogenous jaundice is by far the more common of the two conditions, and the most common cause of this form of jaundice is catarrhal inflammation of the smaller

ducts and common bile-duct which generally occurs in association with gastro-duodenal catarrh.

The following table from Taylor's *Index of Medicine* summarizes the causes of hepatogenous jaundice:

TABULAR VIEW OF THE CAUSES OF HEPATOGENOUS JAUNDICE.

- Obstructive (feces clay-colored)
  - 1. Gall-stones and inspissated bile
    - in common duct.
    - in radicles of ducts.
  - 2. New growth
    - Malignant
      - of liver itself.
      - secondary infiltration of glands in transverse fissure.
      - of stomach.
      - of pylorus.
      - of duodenum.
      - of pancreas.
      - of kidney.
    - Non-malignant
      - syphilis.
      - lymphadenoma.
  - 3. Catarrh of stomach and duodenum.
  - 4. Abdominal aneurism.
  - 5. Hydatid cysts.
  - 6. Accumulation of feces.
  - 7. Ovarian or uterine tumors.
  - 8. Perihepatitis.

As a result of this catarrhal process the bile-duct becomes blocked by the swollen mucous membrane and the mucus which is secreted; the biliary coloring-matter is absorbed into the hepatic circulation and general circulation, and is by this means distributed over the body. Another common cause of hepatogenous jaundice is the obstruction offered to the flow of bile by the presence of a gall-stone or gall-stones in the ducts; and a third cause of obstructive jaundice so-called is pressure on the ducts by growths or inflammatory products in the immediately adjacent organs, or of adherent inflammation in the ducts themselves, or by the presence of a round worm in the duct. Very rarely the jaundice may arise from pressure on the common duct produced by floating kidney.

Catarrhal jaundice of the acute type is generally produced by indiscretions in diet associated with exposure. The patient after more or less marked symptoms of gastric and intestinal disturbance and indigestion feels wretchedly. There is a premonitory mental heaviness, with languor and malaise, and within forty-eight hours or less the yellowing of the conjunctiva and skin appears. The temperature is generally subnormal to a slight degree. The tongue is heavily coated and often somewhat dry. There are marked loss of appetite, great distress, headache, and depression of spirits. Examination

of the hypochondrium may reveal some local tenderness and slight hepatic enlargement, while the abdomen will be in some instances markedly tympanitic as a result of intestinal fermentative processes in the absence of antiseptic bile. The bowels are constipated, often refusing to move except with powerful purgatives. There is little pain, except headache. This condition lasts for a few days or a week, when the color of the skin and conjunctiva usually begins to fade and the normal hue is reached in the course of a week or more. The presence of persistent jaundice should raise the suspicion that it is due to more serious disorder than simple catarrhal inflammation.

The jaundice due to obstruction by stone may be due to blocking of the biliary duct, whereby there is a stagnation of the flow with reabsorption of the bile, or to stoppage of the flow by the presence of a stone in the common duct just as it enters the bowel. A differential diagnosis as to whether the stone is in one or the other of these places is often impossible, but in the variety in which the obstruction is below the opening of the cystic duct it may be possible sometimes to discover by abdominal palpation a pear-like swelling due to a distended gall-bladder.

The jaundice of gall-stone obstruction may be sudden or gradual in onset. If sudden, it is often, but not always, preceded by a violent attack of pain in the hypochondrium, or, in other words, hepatic colic, in which the agony is excruciating and is accompanied by nausea and vomiting. The area of the pain is, however, distinctly hepatic, and it does not radiate down the inside of the thigh and into the testicle or penis as does that due to renal calculus. In place of the subnormal temperature so often seen in catarrhal jaundice we find in obstructive jaundice that the temperature is often considerably raised, and this is particularly apt to be the case in those instances in which the onset is gradual and the jaundice persistent, being due to reflex irritation, or septic absorption, produced by the impacted stone, which may be scratching or ulcerating the lining membrane of the duct. The history of repeated attacks of gall-stone colic, the presence of gall-stones now and then in the stools, the swollen gall-bladder in which, in very thin persons, the stones may sometimes be felt, the age of the patient, who is generally in or past middle life, and the fact that the patient is a female, all point to gall-stone as a cause of the jaundice. As a rule, there is great loss of flesh in all forms of jaundice; but if the local damage done by the stone is great and septic absorption is marked and the fever high, the failure in

strength may be most alarming, while the repeated rigors and sweats increase the distress of the patient.

Jaundice very rarely arises from pressure on the ducts by an aneurism of the abdominal aorta, or more commonly from disease involving the hepatic artery. Three such cases are recorded by Frerichs. Jaundice has also been seen in aneurisms of the superior mesenteric artery as the result of pressure and in cases in which there has been, or is, perihepatitis, with displacement of the liver in such a way that the adhesions cause twisting or dragging on the ducts.

The jaundice of malignant disease pressing upon the gall-ducts is usually not intense, and is characterized by the physical signs of a tumor, by the marked wasting of the patient, and, as a rule, by the very gradual onset of the pigmentation of the skin. Generally the lesion in such cases is carcinoma of the head of the pancreas.

Jaundice is also seen in hepatic hypertrophic cirrhosis to a slight extent in a small proportion of cases, and it is to be remembered that in those cases of this disease in which delirium and muscular twitching occur that the symptoms may resemble acute yellow atrophy of the liver, and all forms of jaundice produce headache and may cause delirium. In acute yellow atrophy of the liver (see below) the liver is greatly reduced in size, whereas in hypertrophy it is greatly increased in size; and in atrophy the temperature is subnormal, whereas in the jaundice due to hypertrophic cirrhosis it is apt to be above normal. Jaundice also may be a manifestation of acute poisoning by phosphorus, which condition is generally accompanied by hepatic swelling and tenderness and with coffee-ground vomiting.

Jaundice is present in all fatal cases of yellow fever and often in cases which ultimately recover. It also is a constant symptom in Weil's disease, which is probably in reality a septic icterus, but it is very rarely seen in suppurative hepatitis. A fleeting and light hue of jaundice is sometimes seen in cases of chronic valvular cardiac disease in which compensation is gradually failing. Rarely this hue becomes deeper as the heart-failure increases. This jaundice is due to engorgement of the liver (nutmeg-liver) which in time results in catarrh of the bile-ducts, with consequent obstruction to the flow of bile.

In amyloid disease of the liver Bartholow states that jaundice occurs in about one-tenth of the cases as a result of enlargement of the lymphatics in the hilus with pressure on the hepatic duct. In

jaundice resulting from cancer of the liver the growth must be so situated as to compress the ducts, consequently jaundice only occurs in about one-third of the cases. Similarly jaundice may result from the presence of echinococci, but this is not a common symptom of the growth of these parasites and the disease is very rare in the United States.

Jaundice sometimes complicates diabetes. Under these circumstances it may be regarded as a coincidence or a valuable diagnostic aid, for, as we have already stated, tumors of the pancreas by pressing on the common duct may cause jaundice, and, as is now well known, widespread disease of the pancreas may cause diabetes. Jaundice in a case of diabetes should therefore direct attention to the pancreas.

In this connection it is well to remember that Hanot, under the name of diabète bronzé, has described one other pigmentation of the skin which contains iron (that of Addison's disease and melanæmia do not), and which is associated with diabetes and hypertrophic cirrhosis of the liver. The coloration occurs most markedly upon the face, limbs, and genital organs; the glycosuria is abundant and slight ascites may be present.

Other noteworthy symptoms of hepatogenous jaundice are intense itching of the skin; a very slow pulse when the patient is at rest, due to stimulation of the vagus by the bile in the blood; and staining of the sweat due to the bile-pigment may also be present. Should the jaundice be due to gall-stones impacted in the ducts, and producing irritation or ulceration of their lining so that septic absorption or "Charcot's fever" develops, the pulse may become more rapid and running, from the general feebleness which rapidly asserts itself. Rigors of extreme severity, followed by sweatings and marked febrile movement, develop in such cases, the chills occurring daily or periodically in a manner closely resembling those of intermittent fever. As these symptoms sometimes develop in cases in which the post-mortem discovers no sign of pus, it has been thought that the disturbances were due to reflex causes, but the opinion of Charcot that there is present in all such cases a true infection seems the most probable. When the gall-stone produces active suppuration the fever becomes more like remittent fever and the patient rapidly emaciates and presents all the signs of active suppuration.

The urine in all cases of hepatogenous jaundice is heavily bile-stained (see Urine), and the stools generally clay-colored, owing to absence of bile in the feces.

A very rare cause of jaundice is that which arises from acute yellow atrophy of the liver, a disease which is seen somewhat more frequently in women than in men, and particularly in association with pregnancy. The age of occurrence is usually between the twentieth and thirtieth year. The symptoms begin with gastro-intestinal disorder, followed by headache, delirium, muscular twitching, and perhaps convulsions. Simultaneously with the onset of the headache the jaundice appears, the patient becomes typhoidal and dies from exhaustion, although recovery has been known to occur. The stools during the attack are clay-colored and the urine contains leucin in discs and tyrosin in needle-like crystals.

Hematogenous jaundice is due, as its name implies, to breaking down of the blood to so great an extent that the liver cannot deal with the waste material with sufficient rapidity, and as a result altered hæmoglobin is deposited in the tissues. Any poison which produces excessive *hemolysis*, such as picric acid and the coal-tar products, chlorate of potassium, glycerine, and poisonous mushrooms, may cause this condition to develop, and in extreme malarial disease (remittent and pernicious malarial fever), dengue, relapsing fever, pernicious anæmia, pneumonia, and in other infectious maladies jaundice may be produced in this manner. It is particularly apt to occur in cases of marked sepsis.

Its causes are shown in the following table from Taylor's *Index* :

CAUSES OF HEMATOGENOUS JAUNDICE.

- Non-obstructive (feces normal in color)
  - Fevers
    - Yellow fever.
    - Typhus fever.
    - Scarlet fever
    - Relapsing fever.
  - Poisons
    - Animal
      - Snake-bite.
      - Pyæmia.
    - Chemical
      - Phosphorus, copper, mercury, antimony, chloroform, ether.
  - Acute atrophy of liver.
  - Neuroses: Joy, grief, fear, passion.
  - Cirrhosis of liver in its latter stages.

Jaundice sometimes occurs after severe hemorrhage of a prolonged character and in prolonged exhausting fevers, and is then due not to any local hepatic trouble but to blood-changes, with the production of urobilin in excessive amounts. The urine fails to carry off all the urobilin which is produced from hæmatoidin or bilirubin. This condition is called "urobilin icterus."

In nearly all cases of hematogenous jaundice the discoloration of the skin is very slight, and the important fact is to be remembered that the stools are not light or clay-colored as in hepatic jaundice, but contain a normal or excessive amount of pigment. Again, the systemic symptoms of catarrhal or obstructive hepatic jaundice are practically absent in the hematogenous variety, and the jaundice is simply a minor symptom associated with the more grave manifestations which characterize the individual infectious process. If the poisoning is very marked, convulsions, coma, or active delirium may come on, but it is probable that these symptoms are due more to the poison of the disease than to the broken-down blood.

Vierordt states that a very small amount of biliary coloring-matter is often found in the urine of patients suffering from pyæmic jaundice, and regards this as an important sign that the discoloration of the skin is due in a given case to blood-changes and not to biliary obstruction, whereas an excessive amount of biliary matter in the urine indicates hepatic trouble.

There still remains to be considered the jaundice seen in the newborn, usually within the first or second day of life (icterus neonatorum), which some believe to be due to a decrease in the blood-pressure in the portal vessels, subsequent to the arrest of the placental circulation, with consequent absorption of bile into the blood, owing to the comparatively high tension of this fluid in the bile capillaries. Others think this jaundice is due to breaking down of the blood-corpuscles shortly after birth as the result of some mild infection. Probably both causes act in some cases. If the cause be the altered blood-pressure, the progress is favorable, and recovery takes place in about ten days or two weeks; but if the cause be an infection, the condition often rapidly proves fatal. Should the jaundice of the newborn be very marked the patient may be suffering from congenital stenosis, or absence of the common or hepatic duct (which cause is rare); from septicæmia, through infection by way of the umbilicus; from phlebitis of the umbilical vein, or from a hepatitis due to hereditary syphilis. In any of these latter causes death will probably occur, whereas in the mild form of jaundice of the newborn the prognosis is very favorable, even though the discoloration lasts for weeks. The mild form of icterus neonatorum, if due to blood-changes, is rarely accompanied by great discoloration of the urine, and the feces are usually no lighter than normal in color;

but if hepatic disease be present, the urine is bile-stained and the feces are light in hue.

Jaundice sometimes comes on in the course of acute ulcerative endocarditis and has been mistaken for that of acute yellow atrophy of the liver, and it often appears as a symptom of pernicious malarial fever, with vomiting, diarrhœa, and grave nervous symptoms.

Rarely jaundice follows severe fright or extreme anger, and Da Costa states that it sometimes ensues after concussion of the brain.

Other Changes in the Color of the Skin. A condition of the skin characterized by yellow, more or less elevated, patches is xanthoma, which Murchison states often complicates hepatic trouble, and which in its nodular form may possibly attack the liver and so produce jaundice. Its favorite distribution is about the eyelids, but it may appear elsewhere. Similar lesions to xanthoma sometimes appear in the course of diabetes (Hutchinson, Besnier), and under these circumstances generally develop suddenly, and spontaneously disappear after some weeks or months.

When the skin of the entire body, the face being particularly affected, is of a livid or bluish-slate color, resembling somewhat the appearance of a person exposed to rays of light passing through blue glass, the condition is that of argyria or chronic silver-poisoning. This discoloration is so characteristic as to admit of no difficulty in diagnosis, since the absence of any circulatory or respiratory embarrassment excludes the possibility of its being due to cyanosis. Owing to the decrease in the amount of silver given internally by physicians, chronic argyria is becoming more and more rare. The discoloration is due to a deposit of oxide of silver in the rete Malpighii.

Discoloration of the skin of the entire body of a sallow, lemon-yellow tint, sometimes called a "muddy yellow" hue, is seen in persons who are sufferers from prolonged malarial poisoning, and in some cases the subject of prolonged suppurative processes not tubercular in character. A greasy, yellowish skin does, however, occur as an accompaniment of some cases of pulmonary phthisis, and these cases have as a rule a gloomy prognosis. Often chronic hepatic disease, such as cirrhosis, produces this sallow appearance.

Other changes in the color of the skin, which cannot be said to be due to deposition of pigment, although they seem to be caused by this, are seen most markedly in the peculiar yellowish cheesy pallor of carcinoma, the greenish-yellow tinge of true chlorosis, the

curious cadaveric hue of advanced pyæmia, and the yellow skin with a greasy feeling in some cases of paretic dementia.

Local pigmentations of the skin result from many causes, both local and systemic, direct and indirect. When brownish-yellow spots or streaks appear on the face, so that chloasma is developed, we should look for uterine or hepatic disturbance or pregnancy; they are practically large freckles of a more or less distinct brown hue. In other instances, chloasmic spots or localized discoloration of the skin results from injury to the skin, as pressure by clothes, chafing, or after constant severe scratching in the course of eczema or pediculosis or scabies. If the pigment is found in the nuchal and sacral region, it is probably from the scratching caused by pediculi; if on the body in irregular distribution, it may have been caused by prurigo. Again, the presence of a brown pigmentation of the skin in clearly outlined patches may indicate the earlier use of a fly-blister, a mustard plaster, or other counter-irritants, and a brown discoloration of the skin, which might possibly be confused with that of Addison's disease, is produced by the free use externally of oil of cade.

Sometimes these spots are produced by the prolonged use of arsenic, and the writer has reported a case in which the coalescence of the spots produced a curious grayish-brown hue of the entire body, so that the man looked somewhat like a mulatto.

Sometimes brown pigmentation of the skin of the neck and face appears as a symptom in exophthalmic goitre, and this disease may also produce similar lesions on the chest and wrists. Very closely resembling these spots is the bronzing of the skin in patches which is seen in persons suffering from Addison's disease; but although bronzing of the skin is a somewhat constant symptom of Addison's disease, its presence is neither a positive nor negative sign in diagnosis, for bronzing is sometimes seen in cases in which the suprarenal capsules are normal. In some instances the bronze color deepens into a dark gray or even a black hue, and although the discoloration is generally in patches, it may extend over the entire surface of the skin, even to the edges of the finger-nails. The nails, however, escape, as does also the mucous membrane of the lips, although the lining of the mouth itself may be dotted with pigmentation. The color is due to pigmentation of the rete Malpighii, and pressure has no effect on it. The symptoms of Addison's disease to be found associated with these skin-changes are "anæmia, gen-

eral languor or debility, remarkable feebleness of the heart's action, and irritability of the stomach." (Addison.)

The slate-colored skin of argyria or chronic silver-poisoning can be readily distinguished from the bronze color of Addison's disease; but if a further test is needed, it will be found that washing the skin of argyria with a solution of iodine changes its color, while that of Addison's disease remains unaltered.

White patches, or leucoderma, are also sometimes seen in some cases of true goitre, and brown ones in tuberculosis.

In carcinoma of one of the internal organs, or of the breast, of an advanced form, the appearance of the skin is drawn and unusually smooth, often shiny or greasy-loooking, somewhat gummy and leathery to the touch, particularly where the integment is naturally dense. Although it is difficult to describe, this skin is almost pathognomonic of carcinoma, although it may also be present to some extent in far-advanced cases of pernicious anæmia or sarcoma.

Pallor of the skin is due to absence of the normal pigment, to deficient blood, to central or local vasomotor disturbance as is typified by fainting, and far more rarely by Raynaud's disease. As a type of the pallor due to lack of pigment in the skin we see vitiligo, while the pallor due to pernicious anæmia or pseudo-leukæmia and malaria is due to lack of red corpuscles. Similarly, a pallor due to lack of hæmoglobin is typified by chlorosis. (See Blood.) In all of these diseases the pallor of the skin may be of ghastly whiteness or tinged with yellow. The skin is apt also to be very white, and even chalky in appearance, in chronic contracted kidney and chronic parenchymatous nephritis.

In chlorosis the entire surface of the body is exceedingly pale, and the skin of the face, particularly about the mouth and nose and eyes, is somewhat greenish in hue. A very important diagnostic point to be remembered is that red cheeks often cause the physician to overlook well-advanced anæmia in young women. (See chapter on the Blood.)

In those cases in which the skin is pale from alteration of the subcutaneous circulation there is usually incompetence of the heart or vasomotor disturbance, but the most marked form of general pallor is that due to myxœdema.

Cyanosis or blueness of the skin depends upon the circulation in the subcutaneous vessels of imperfectly oxidized blood The small veins are often seen to be swollen, particularly those of the face and

the hands and feet. The most marked form of cyanosis with which we meet is the cyanosis of the newborn child, suffering from a patulous foramen ovale, and in this condition the color may vary from slate-colored blue to an almost black hue. The lobes of the ears, the tongue, the scrotum, and the toes show the color most deeply. It is important to remember that this form of cyanosis is greatly decreased as a rule by placing the child on its right side. Anything which produces excitement increases the cyanosis greatly, whereas cyanosis due to other causes is not subject to great variations. In the cyanosis of the newly born, males are far more frequently affected than females, in the proportion of about 2 to 1 or 3 to 1, and it is a noteworthy fact that even when the cyanosis is due to a malformation of the heart it may not be present from the time of birth, but may develop several days afterward. J. Lewis Smith records forty-one cases in which the cyanosis due to a congenital heart-lesion came on at periods ranging from two weeks to forty years after birth.

About 35 per cent. of the cases of cyanosis due to congenital defects die in the first year. The following table from Lewis Smith shows the character and relative frequency of these lesions :

	Cases.
1. Pulmonary artery absent, rudimentary, impervious, or partially obstructed	97
2. Right auriculo-ventricular orifice impervious or contracted	5
3. Orifice of the pulmonary artery and the right auriculo-ventricular aperture impervious or contracted	6
4. Right ventricle divided into two cavities by a supernumerary septum	11
5. One auricle and one ventricle	12
6. Two auricles and one ventricle	4
7. A single auriculo ventricular opening; interauricular and intraventricular septum incomplete	1
8. Mitral orifice closed or contracted	3
9. Aorta absent, rudimentary, impervious, or partially obstructed	3
10. Aortic and the left auriculo-ventricular orifice impervious or contracted	1
11. Aorta and pulmonary artery transposed	14
12. The cavæ entering the left auricle	1
13. Pulmonary veins opening into the right auricle or into the cavæ or azygos veins	2
14. Aorta impervious or contracted above its point of union with the ductus arteriosus; pulmonary artery wholly or in part supplying blood to the descending aorta through the ductus arteriosus	2
Total	162

The chances are about ten to one that in cyanosis of the newborn the lesion is absence of a properly developed interauricular or interventricular wall.

In the adult or child cyanosis may be produced by serious cardiac disease, by pulmonary disease, such as pneumonia, pul-

monary congestion, and bronchiectasis with emphysema and associated cardiac dilatation. It also occurs in laryngeal obstruction arising from external pressure or intralaryngeal difficulty, and in cases of asthma of a severe form. (See chapter on the Thorax and its Viscera.)

In some cases of paretic dementia the skin of the forehead is dull and dusky-looking. In other instances a grayish-blue or cyanotic appearance may arise from the ingestion of drugs, which reduce the hæmoglobin of the blood, such as antipyrine or acetanilid, and in such instances the discoloration is first seen about the base of the thumb-nail or in the skin of the face, particularly if the patient be examined from a little distance.

The condition of the skin, so far as its nutrition is concerned, is of great importance in diagnosis. In profound failure of the vital forces continuing over a great length of time it becomes abnormally dry and scaly, the hair becomes straggling and lustreless and frequently falls. In young persons suffering from grave disease of the lungs or heart of a chronic type there is often not only an undue dryness of the cuticle, but an abnormal growth of downy hair all over the body and limbs, and more particularly down the spine and over the breast-bone.

Eruptions on the Skin. The influence of age upon the development of skin lesions is very great, and Stephen Mackenzie has summed up the relationship of skin disease to age in the following amusing manner: "The seven stages of man could be well illustrated by diseases of the skin, though we lack a Shakespeare to do justice to the theme. In the 'mewling and puking' infant we meet with sclerema and œdema neonatorum, the 'red gum' or strophulus of the older writers, intertrigo, eczema, urticaria papillosa (lichen urticatus), urticaria pigmentosa, xeroderma pigmentosum, and impetigo; the 'schoolboy,' with his chilblains and ringworm, alopecia areata, pityriasis rosea, ecthyma, and 'football disease;' and then the 'lover,' with his acne and sycosis, and, as a result of irregular sexual excursions, his syphilides; 'and then the justice, in fair round belly' with acne rosacea, diabetic boils, and pruritus ani; the sixth stage shifts into the 'lean and slippered pantaloon,' with rodent ulcer and 'gouty' eczema; 'last scene of all, sans teeth, sans eyes, sans taste, sans everything'—except an incessant and intolerable itching of the skin which we call senile prurigo."

There are two conditions of the skin in which valuable evidence is given that the patient is suffering from rheumatism. One is the presence of erythema in one of its many forms, the other is the appearance of purpura, or, as it has been called, peliosis rheumatica. That the presence of erythema points in many cases to rheumatic trouble is proved beyond all doubt, either erythema papulatum, annulaire, marginatum, or nodosum being indicative of the systemic taint, but it is worthy of note that the erythema marginatum is most diagnostic and erythema nodosum the least diagnostic of rheumatic poisoning. Sometimes this eruption may be the only manifestation other than cardiac involvement, and when the marginate eruption is present severe cardiac involvement is commonly seen. The papulate eruption is most commonly found on the back of the wrists, the hands, and the feet when it occurs as a rheumatic sign, while the nodose variety is generally confined to the front aspect of the legs or the extensor surfaces of the arms. It must be remembered that these forms of erythema may be distributed anywhere over the body in rheumatism, but that they become especially diagnostic if limited to the areas named.

Purpuric discolorations of the skin, somewhat resembling multiple bruises in appearance, are due to a number of causes and possess a varied significance. In the first place, they are due to the disease known as purpura hæmorrhagica, which may be divided into the acute and subacute forms, and that which is secondary as the result of severe infections and certain poisonings. The acute form of purpura runs a rapid course and reaches a fatal result in most cases in a short time. It is a comparatively rare disease and usually attacks young adults, chiefly males, up to twenty-eight years of age. It is sometimes seen in young girls and more rarely in young pregnant women. The chief symptoms consist in hemorrhages from the mucous membranes, purpuric spots, high fever, and a general class of symptoms resembling those of sepsis, as chills, pyrexia, and exhaustion. In other instances active hemorrhages take place into the viscera, and if into the meninges of the brain cause cerebral symptoms at once. The liver and spleen are nearly always enlarged.

The subacute type, while severe, runs a far more favorable course as to its manifestations and results. It usually attacks children or young adult males from twenty to thirty years of age. The patient after a feeling of wretchedness, and perhaps a chill, followed by the

purpuric eruption, is attacked by swelling of the joints and perhaps hemorrhages from the kidneys, bowels, and mucous membranes. If the hemorrhage be from the gums, the teeth are not loosened, as in scurvy. Prostration may be great and the patient appear as if suffering from typhoid fever. The prognosis is good for ultimate recovery. It is sometimes called peliosis rheumatica or Schönlein's disease. This subacute form, however, occurs in a more severe manner, as "Henoch's disease," in the persons of children between nine and twelve years, and is much more common in males than females (five to one). In this form we have as additional symptoms marked pain and tenderness in the belly and bloody stools, with tenesmus and active vomiting. The illness may last a long time, but recovery often occurs, about 25 per cent. dying. The joint-symptoms of the other forms of purpura may be slight or absent. Often, too, the purpura is accompanied or replaced by erythema.

Subcutaneous fibroid nodules sometimes occur in cases of rheumatism and vary in size from a hemp-seed to a walnut. They are usually situated in the subcutaneous connective tissue, but may be attached to the deep fascia or muscular sheaths.

The question as to whether purpuric eruptions are ever truly indicative of rheumatism has been much discussed and their diagnostic value denied, but the author believes that in some cases of rheumatism purpura is a symptom, appearing often in the neighborhood of the involved joints, nearly always on the lower limbs, and often breaking out before any evidence of articular trouble exists. In other instances the development of the purpura is simultaneous with the disappearance of joint-trouble. The eruption usually fades in a few days, but frequent relapses or new crops of it often occur.

Purpuric eruptions may be produced by quinine in persons who have an idiosyncrasy to this drug and by iodide of potassium, chloral, and salicylic acid. They may also accompany any severe infectious disease and follow the entrance into the body of any poison which destroys the blood, such as the poison of snake-bite. They also result from severe jaundice, from profound anæmia, from congenital syphilis with vascular changes, from endocarditis (a form of sepsis), and in cases of multiple sarcomata. Rarely purpura has followed fright and severe grief.

Urticaria may occur as a manifestation of rheumatism, but it has no diagnostic value. Sometimes it ensues upon the use of salicylic acid, turpentine, and quite commonly follows the ingestion of iodide

of potassium. The wheals produced by the latter drug differ from those of urticaria by being unduly red.

The development of polymorphic skin lesions, consisting of hyperæmia, œdema, and hemorrhage, with arthritis occasionally and visceral disturbances, consisting in attacks of vomiting or diarrhœa, endocarditis, pericarditis, acute nephritis, and hemorrhages from the mucous membranes, indicates the presence of a condition called erythema exudativum multiforme. The attacks are apt to be recurrent. Sometimes the skin-manifestations are absent.

Hemorrhages of the skin occur spontaneously in some cases of hysteria and paretic dementia and after epileptic attacks, particularly about the eyes, and often from injuries received in other parts of the body during the convulsion. Minute hemorrhages may also occur in the course of severe whooping-cough, and, in the form of petechiæ, result from snake-poisoning, septicæmia, cerebro-spinal meningitis, iodism, ergotism, and after inhaling the vapor of benzine. They are also seen in scurvy and in some cases of profound wasting, as in the course of phthisis and carcinoma.

Petechial rashes closely resembling those of malignant smallpox, typhoid fever, or cerebro-spinal fever may be due to the presence of acute ulcerative endocarditis.

Hemorrhages into the skin sometimes also appear in the skin covering a part affected by a severe pain in the crisis of locomotor ataxia.

A very extraordinary manifestation of spontaneous subcutaneous hemorrhage is seen in what is known as hæmatoma auris, a condition in which a free extravasation of blood takes place into and beneath the skin of the ear. The color of the swollen ear is quite red in color if the hemorrhage has been recent, or dark blue if it is an old occurrence. The left ear is more commonly affected than the right and it is seen more commonly in males than females.

Excessive redness of the skin is seen in acute inflammations of the skin or the subcutaneous tissues, and as the result of hot applications, the redness being more and more marked as the heat is prolonged and is great. Often the prolonged use of great heat will produce a peculiar mottling of the skin like that of an old bruise.

Aside from the redness of the cheeks and forehead from blushing we should remember the general flushing seen so commonly in persons suffering from phthisis, particularly when they are excited, which differs from the more dusky redness seen over the malar bones in hectic fever.

Another interesting diagnostic sign in the skin is what is known as the "*tache cerebrale*," a condition of vasomotor disorder in which when the finger is gently drawn over the skin of the forehead a red patch speedily develops. It is seen in meningeal irritation, brain abscess, epilepsy, in some cases of exophthalmic goitre, and in paretic dementia. Sometimes it is called "*tache meningeale*."

Erythema or rose-rash, sometimes called roseola, is a redness of the skin and occurs in many pathological conditions. It may be localized or diffused. In a number of diseases it aids us very greatly in reaching a diagnosis, but the physician should always be cautious in depending much upon it, since it may mislead, owing to the fact that it often appears when devoid of diagnostic importance.

The development of a diffuse, punctuated rose-rash on the skin of a person who is suffering from malaise, fever, nervous disturbance, and sore throat should direct the physician's attention to the possible presence of two infectious diseases, namely, scarlet fever, which is more common in childhood, and syphilis, which is more frequent in adults. The rash of scarlet fever is of a very bright-red color and shows itself at the end of the first, or on the second day of the disease, first appears on the chest and neck and then speedily involves nearly the whole surface of the body, although the forehead often escapes and the skin about the corners of the mouth remains nearly always white and free from eruption. On the other hand, the soles of the feet and palms of the hands are very markedly affected. So intensely reddened is the patient's surface that it may have the color of a boiled lobster. This redness depends upon an acute hyperæmia of the skin, which though removed by pressure instantly returns when the finger is withdrawn. A noteworthy point is its punctate and mottled appearance, for, while the entire skin may be red, there are points which are more red than the rest of the skin and also certain areas which are particularly so. The skin is often slightly swollen and feels tense, and itching is commonly present. The rash usually lasts three or four days and then fades, desquamation of the cuticle speedily setting in, which is completed in about two weeks. Sometimes, however, it remains for ten days to three weeks. Often when the rash can scarcely be seen on the skin its full development will be found on the pharyngeal wall. In the malignant types of scarlet fever petechiæ and subdermal hemorrhages occur.

An erythema resembling scarlet fever, not only in its appearance,

but also by its association with swelling of the lymphatic glands and reddening of the mucous membranes of the mouth, sometimes develops about the second or third day in cases of dengue or break-bone fever.

In children there are several other conditions than scarlet fever which are associated with rose-rash, and these are more apt to lead to errors in diagnosis than is the rash of syphilis. The most frequent of these is erythema roseola, or roseola of acute indigestion, or that following the use of a food to which the patient has an idiosyncrasy. It is generally, but not always, widely diffused and is often associated with acute and severe febrile movement and vomiting, but it can be separated from scarlet fever by the facts that there is an absence of severe constitutional and nervous symptoms (except in neurotic children), there is no sore throat nor enlarged cervical glands, and the rash does not come out on the clavicles and gradually travel down the body.

Another condition closely resembling scarlet fever is rarely seen, namely, acute exfoliating dermatitis, called, in its mild form, *erythema scarlatiniform*, which has a sudden onset with febrile movement and a rash which rapidly spreads over the entire body and lasts four or five days, finally ending in desquamation. So closely may this disease resemble scarlet fever that a diagnosis during the first attack may be impossible for the first few days, but the condition of the throat and tongue does not resemble the condition seen in scarlatina. Desquamation is often even more complete than in scarlatina, and the hair and nails are frequently shed. Relapses are very common and give rise to the reported cases of repeated attacks of scarlet fever.

The rash of rubella or rötheln (German measles) is a roseola and more closely resembles that of scarlet fever in some cases than it does that of measles, but it is never as scarlet, is distinctly maculated, and only at a distance looks homogeneous. Close examination always reveals the rash in oval patches, and it lacks the diffused character of the rash, the punctation of the skin, the grave systemic disturbance, and the throat-symptoms of scarlet fever. Further, the febrile movement is comparatively slight and the rash lasts only two or three days. Slight desquamation may, however, occur.

The eruption of measles (morbilli or rubeola) is very characteristic and can be in most cases easily separated from the other exanthemata by close examination. It is a roseola in character, but more dusky than that of scarlet fever. It appears about the fourth day of the

illness in association with catarrh of the mucous membrane of the eyes and respiratory tract. Unlike scarlet fever it appears in macules first upon the forehead or face, then on the neck, trunk, and limbs. The macules, which often coalesce, are arranged in crescents which are red, but become somewhat yellowish on pressure. They are slightly raised. There is nearly always to be seen some uninvolved skin, the entire surface not being covered as in scarlet fever. In some instances in which the eruption is aberrant a diagnosis of measles from scarlet fever is admittedly impossible until the case has been watched for some days, but the slow onset of measles, in which the eruption appears on the fourth day as against the first day in scarlet fever, the swollen eyes and nose, the puffiness of the face, the catarrhal condition of the mucous membranes, the curious fall of temperature after the preliminary rise on the first day, the short duration of the rash, all aid in the diagnosis of measles. The dusky eruption of measles can nearly always be found on the pharyngeal mucous membrane.

Roseola sometimes appears after vaccination, and in cases of smallpox previous to the outbreak of the true eruption. Under the latter circumstances it is found most commonly about the groin and inner surface of the thighs and on the hypogastrium, loins, clavicles, and the extensor surfaces. So closely may the early rash of smallpox simulate the aberrant types of measles as to lead to grave mistakes in diagnosis. Sometimes an immediate diagnosis is impossible, even by the most experienced, but the rash of measles commonly appears on the face, therefore this difference, coupled with a history of exposure, the gradual development of the peculiar "shot under the skin" sensation of variola, and the ultimate distinct papulation, vesiculation, and pustulation of smallpox soon remove the doubt in the physician's mind.

DAY OF ERUPTION OF THE VARIOUS EXANTHEMATA.

Day.	Disease.	Area.
First to second day	Rötheln or German measles. Varicella or chickenpox.	Face first. Face or trunk.
Second day	Scarlet fever.	Neck and chest.
Third to fourth day	Measles or morbilli or rubeola. Variola or smallpox.	Face. Forehead, face, and wrists.
Fourth to fifth day	Typhus or ship fever.	Trunk.
Seventh to ninth day	Typhoid or enteric fever.	Abdomen.

A diagnosis between the eruption of measles and variola often can be made by stretching the skin between the fingers, when, if it be measles, the papule cannot be felt, whereas, if it be variola, it persists. This is called the "grisolle sign."

Among other diseases in which rose-rash appears we find diphtheria, septicæmia, cholera, typhoid fever, malarial poisoning, and Bright's disease. In diphtheria it may lead the physician to a diagnosis of scarlet fever with severe faucial manifestations, and only a careful examination of the throat, the rapid subsidence of the rash, and the bacteriological examination of the false membrane will settle the diagnosis. Sometimes, however, a roseola appears late in the course of diphtheria, probably as a result of septic absorption. The presence of a very high temperature, of nervous irritability, and the predominance of the throat-lesions of scarlet fever ought to decide the diagnosis in favor of scarlet fever.

The roseola of early syphilis resembles that of scarlet fever, in that it first appears on the trunk, but it is not bright scarlet, but rather dusky red. It appears in patches and is not diffuse, and it ensues about six weeks or three months after the appearance of an initial lesion, occurs in an adult as a rule, is not associated with high fever, and soon involves the face and forehead. These symptoms aid us in separating it from scarlet fever, although it often appears in full blast in the palms of the hands and soles of the feet, but a roseolous rash in these areas in an adult is always suspicious of specific trouble. These patches speedily change from rose-rash to other more marked lesions in cases of syphilis, and one of the first changes that they undergo is to become circinate. They fade and reappear, last an indefinite time, fade in the centre, and so change into marginate or circinate erythema.

When roseola develops after a surgical operation or after delivery in a puerperal female, it is not scarlet fever, but is due to sepsis, although it is of course possible for scarlet fever to attack such cases at any time. The rash is usually found over the abdomen and inner sides of the thigh. The absence of sore throat, the possible presence of a septic process, and the absence of a strawberry-tongue all help to exclude scarlatina. Sometimes, late in an attack of cholera, a rash like surgical roseola appears in the same areas, or in the period of reaction, comes out on the forearms, backs of the hands, and rarely on the back.

The roseolous rash of typhoid is sometimes widely distributed and

almost like measles in appearance, but, as a rule, it is limited to a few or many rose-spots on the abdomen, chest, or back. These rose-spots disappear on light pressure, but immediately return when the pressure is removed, and are most marked in typhoid fever about the seventh to the tenth day of the disease. They may become slightly papular. In the relapse of typhoid fever the rose-spots often appear as early as the third or fourth day. In typhus fever they are much more plentiful and often form petechiæ.

In Bright's disease a roseola often appears over the feet and ankles, wrists, and hands, and sometimes spreads to the skin of the chest and abdomen. Desquamation may take place, but absence of febrile movement and the presence of renal trouble render the diagnosis easy. This manifestation has not a dangerous import.

A dusky red rash rapidly spreading over the neighboring skin, above the level of which the affected area is raised, and which is separated from the sound skin by a sharp line of demarcation which can be both seen and felt, is characteristic of erysipelas. The skin soon becomes brawny to the sight and touch, and the line of demarcation feels particularly indurated. Most commonly the disease appears on the face, starting from the inner canthus of the eye, the nostril, or the corner of the mouth. Very rarely does erysipelas affect the skin of the trunk. The fever may be quite marked, even in mild cases, and usually falls by crisis on the sixth day. In severe cases with fatal tendencies there may develop in place of crisis a typhoid state with low fever and delirium. If the disease be severe, blebs and bullæ form, and œdema of the skin becomes very profound, and finally suppuration may occur, forming what is known as phlegmonous erysipelas (see also Glanders).

Erysipelatous inflammation of the skin without systemic disturbance may follow the application of arnica. A condition also closely resembling erysipelas in its raised surface is urticaria, which, however, differs so materially in other respects that a diagnosis is readily made. Aside from the absence of systemic disturbance in urticaria the swelling of the skin is not red, but pale and pearly in hue, although it may be surrounded by an erythematous blush ; the onset is extraordinarily sudden, so that a skin seemingly normal at one moment after a slight bruising by the finger or rubbing by the clothes develops the complete eruption in a moment.

A marked roseola or dermatitis involving the inside of the thighs or the scrotum or vulva should give rise to the belief that the patient

is suffering from some failure properly to pass or retain the urine, which, on escaping, irritates the skin. This is particularly apt to result if the urine is that of a diabetic. Again, it is an interesting fact that in some cases of tubercular peritonitis an erythematous rash appears on the abdominal wall around the navel.

The presence of a roseola or erythematous rash often indicates the untoward influence of some drug, following its external or internal use. We find that it very commonly follows the ingestion of copaiba, and, as many persons suffering from venereal disease take this drug, the physician must use care not to be led into a diagnosis of syphilitic roseola. It also follows the use of quinine, opium, antipyrine, and many other drugs, such as digitalis and chloral.

The roseola caused by the use of copaiba appears by preference on the upper and lower extremities and particularly on the backs of the hands, about the knees, the ankles, and on the chest, and it is often accompanied by fever. Indeed, the eruption caused by copaiba may closely resemble a papular syphilide, but its sudden onset, itching, and disappearance when the drug is stopped separate it diagnostically from the specific disease.

The roseola following the use of bromide of potassium is, according to Veiel, very rare, and is distributed over the lower limbs. In children it may closely resemble measles.

The roseola or erythema caused by quinine is to be separated from that of scarlet fever by the absence of fever, of the scarlet tongue and sore throat, and by the fact that there are no prodromes or circulatory disturbance except the characteristic evidence of cinchonism. In doubtful cases this is still further confirmed by analysis of the urine, or by the use of the following simple test. Observe the disappearance of the fluorescence of the urine caused by quinine, after the sodium chloride has been removed by precipitation by nitrate of mercury, or after separating the quinine as an iodide by the addition to the urine of a solution of two parts iodine, one part of iodide of potassium, and forty parts of water. The iodide of quinine can be again dissolved by the application of heat.

A distinct diffuse roseola sometimes follows the use of arsenic.

Roseola may be caused by the use of salicylic acid and strychnine, and a scarlatiniform rash sometimes appears in blotches over the face and body in persons who are taking turpentine.

Roseola also ensues in some persons after the application of surgical dressings containing iodoform, corrosive sublimate, and carbolic

acid, being due either to a local effect of these drugs or to their absorption from the dressings. Arnica tincture applied for sprains or bruises may produce marked roseola, or even erythematous and erysipelatous swelling of the skin as already stated.

By far the most important drug-exanthem is that caused by atropine, the rash produced by it being very like that of scarlet fever, except that it lacks the red punctations of that disease. It may be associated with a slight rise in temperature and be followed, rarely, by desquamation. The face of a child suffering from an overdose of atropine is very characteristic. The eyes are bright, the pupils widely dilated, and the skin over the malar bones is red, but striking lines of pallor reach from the corners of the mouth to the nose. There may be active, talkative delirium and very mild convulsions from atropine in overdoses, thus making the resemblance to the onset of scarlet fever very striking. The brief duration of the rash, its lack of punctation, the absence of high fever, and the history of the patient having taken atropine or belladonna, all help to make the differential diagnosis.

Roseola, followed by desquamation, has been known to follow the hypodermic injection of mercury. Sometimes the use of blue ointment produces a widespread rash resembling measles, and this resemblance may be increased by the development of a febrile movement. A similar eruption may ensue from the ingestion of opium.

Erythematous rashes, too, frequently follow slight irritation of the skin in persons who use chloral.

Acne of the skin, particularly on the face, is often produced by the use of bromide or iodide of potassium, or of any preparation containing bromine or iodine. That produced by iodine is generally sudden in its onset and profuse in its distribution. The base of the pimple is bright red, the top speedily becomes pustular, and Fournier states that it may be hemorrhagic. Stopping the ingestion of the drug speedily relieves, or at least decreases, the eruption. The acne due to bromine is often very profuse, and the pimples in severe cases may coalesce, making sloughs of considerable size with an indurated base.

In some persons, generally females, there is developed an acne on the face, breast, and back, as the result of taking iron as a tonic.

In addition to the acne caused by drugs, or their compounds, we should also mention the acne and furuncles appearing in persons working in paraffin, which is due to blocking of the sebaceous glands.

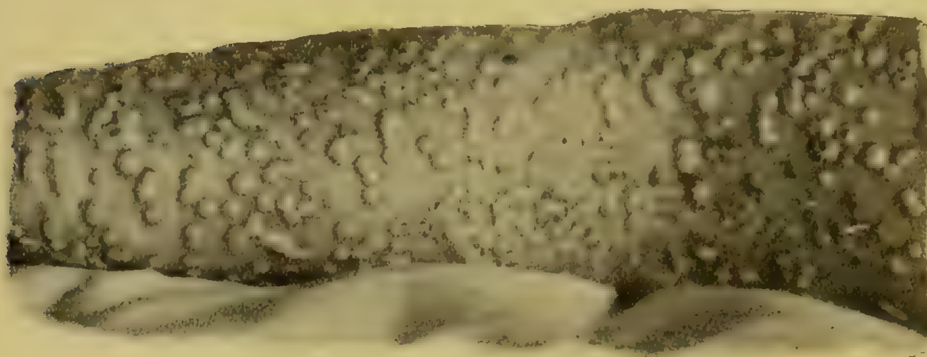
Closely associated with this form of eruption is that which is characteristic of smallpox and chickenpox. The eruption of smallpox appears on the second or third day in the form of tiny specks, resembling flea-bites. These rapidly become papules, which have an indurated base, so that they feel as if shot were under the skin. (Fig. 86.) After about thirty-six hours these papules become vesi-

Fig. 86.

Smallpox eruption on the seventh day.

cles, containing a turbid fluid, which speedily becomes purulent, forming a pustule. (Figs. 87 and 88.) Generally this process of maturation takes three days and, with the development of the pus, the so-called secondary fever, which may be even higher than the primary fever of invasion, sets in. After a period of eighteen to twenty-one days the pustules drop off, having become dried up, leav-

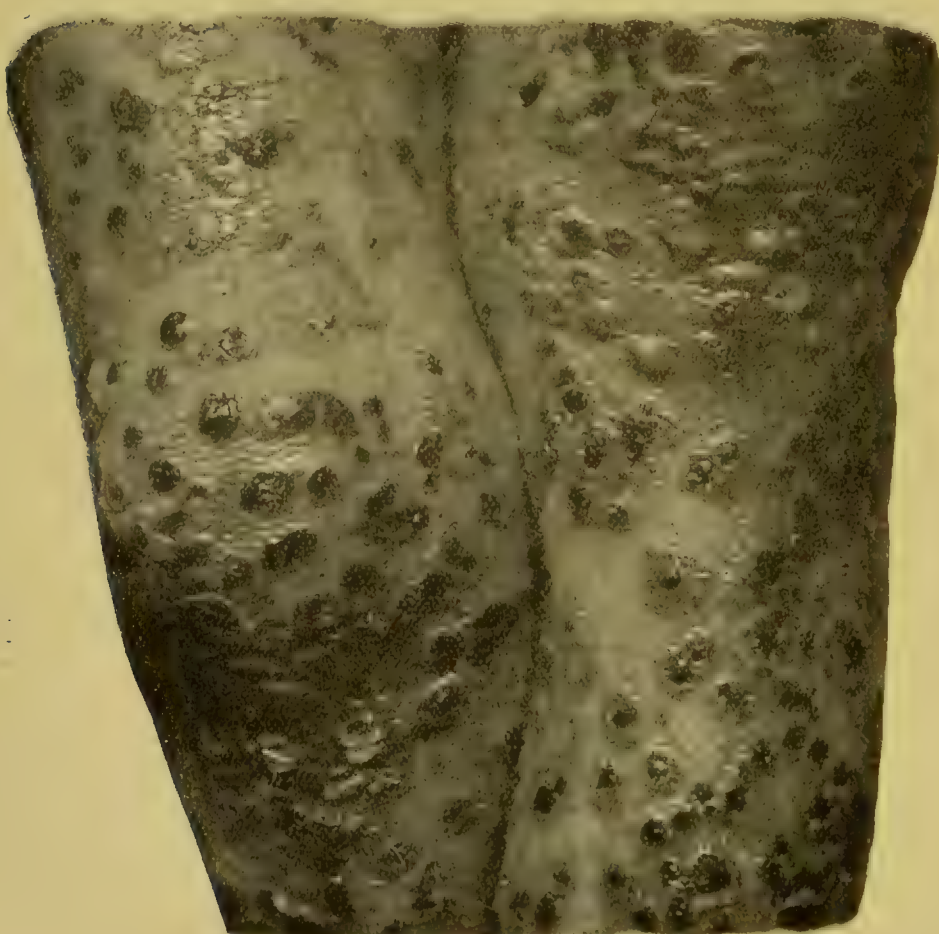
FIG. 87.



Smallpox eruption on the eighth day.

ing, if the attack has been severe, or the skin delicate, deeply pitted scars. Although the eruption of smallpox appears on the forehead,

FIG. 88.



Smallpox eruption on the eleventh day. The pock is seen to be umbilicated.

which is the favorite seat of acne in many cases, a differential diagnosis is not difficult, since the grave systemic disturbance, febrile movement, and rapid involvement of the whole surface of the body speedily indicates the true nature of the disease. The early appearance of the rash on the hands in variola also is a diagnostic sign, as acne in this part of the skin is practically unknown. Then the sudden development of the eruption in smallpox is entirely different from the gradual onset even of the most intense acne. In some cases a purulent acne of the forehead develops in syphilis.

FIG. 89.

Typical vaccine vesicles. Tenth day.

The separation of variola from measles has already been discussed, and it is only in the papular stage that the former disease can be confused with the latter, while the reddened mucous membranes and swollen face of the case of measles soon determine the diag-

nosis. The rapid formation of vesicles and the shot-like sensation of the eruption show that the eruption is not measles.

The appearance of the eruption of vaccinia following vaccination must be next described. Three or four days after the vaccination a single or several papules arise on the scarified surface, which by the sixth day are changed into umbilicated vesicles, which soon unite and form one vesicle the size of a five-cent piece. This vesicle finally forms a scab which falls off after the expiration of about three weeks from the inoculation. A "good take" is always surrounded by an areola of rosy red of several inches in width. Rarely severe inflammation and sloughing ensue. (Fig. 89.)

In chickenpox the eruption appears on first and second day and keeps coming out for several days. It is rose-colored and occurs as papules, which immediately become vesicles. They last but four or five days, and are usually associated with very mild febrile disturbance, the child remaining but little indisposed if well cared for and nursed. Unlike smallpox, varicella does not become umbilicated or leave pits in the skin, unless the vesicles are picked at by the finger-nails. Neither do the vesicles become pustules unless infected by picking or the child is in a condition of debility or suffers from struma. Varicella is separated from variola by the absence of severe systemic disturbance, by the rash first appearing on the chest and neck instead of the forehead and hands, by the presence of other cases of the disease in an epidemic, and, finally, by the fact that it attacks children who have been well vaccinated, whereas smallpox does not. The history of exposure is, of course, an important point to be investigated.

An eruption closely resembling chickenpox or smallpox is that called impetigo contagiosa, in which there are found multiple, flattened, or slightly umbilicated roundish or oval vesicles, pustules, or blebs, which form after some days dry yellowish crusts. It occurs in childhood, or early adult life, and is often associated with some degree of fever. The areas involved are the face, neck, buttocks, hands and feet. The lesions of the skin are larger than in chickenpox. As its name indicates, the disease is contagious, and the occurrence of a series of cases in close proximity to one another should not mislead the physician into a diagnosis of variola or varicella. The eruption lasts about two weeks, and Kaposi asserts that swelling of the submaxillary glands is always present. We can further separate impetigo contagiosa from varicella by the localization of its eruption to

one area as a rule, by the fact that the eruption becomes bullous or purulent and by the larger size of the vesicle. From smallpox we can separate it by the absence of severe pain in the back, the grave systemic disturbance and the secondary fever of that disease, accompanied as they are by the smallness of the pox, the peculiar odor of the patient, and the history of exposure to variola.

In the presence of a papular, pustular, or vesicular eruption of the skin it must be remembered that quinine sometimes develops these lesions in susceptible persons. In some instances where it involves the hands it may indicate that a local effect has been produced by working with the drug.

Eczema in its various forms may appear as the result of the use of quinine internally or locally, or of the employment of mercury internally or externally. When it arises from the use of iodide of potassium, which is very rare, it chiefly affects the scalp and scrotum. The development of an eczematous irritation of the skin sometimes follows the use of chloral.

Herpes labialis is a very constant lesion associated with croupous pneumonia, and is an important sign for the separation of epidemic cerebro-spinal meningitis from meningitis due to other causes, as it is not commonly present in the non-epidemic form. It sometimes arises as a result of using salicylic acid.

The development of recurring crops of boils in persons not exposed to paraffin or tar should cause the physician to suspect the presence of diabetes mellitus, or at least of the presence of general debility and particularly of absence of lime salts from the system in the proper quantity. When the ordinary boil passes into a condition of marked induration about its base, with sloughing of the subcutaneous tissue and necrosis of the skin, which becomes perforated by the openings of several sinuses, we have to deal with a carbuncle or anthrax simplex. The disease usually appears on the back of the neck, on the back, or the lip. The systemic disturbance is very great and the exhaustion profound. The skin covering the area involved becomes grayish or bluish-black, and then separates as a large mass, while the subcutaneous tissues come away in shreds. It is a dangerous disease in all persons, but particularly so in those who are already weakened by other diseases or excess.

The development of a painless macule on the skin of the hand or foot, followed by an acutely inflamed papule which itches and is soon changed into a relaxed vesicle containing bloody serum, in

which there is a hard nucleus which rests upon an indurated base, is the initial manifestation of anthrax maligna or malignant pustule. The lymphatics soon become swollen and metastatic abscesses speedily form elsewhere, as in the axillary glands. The systemic symptoms are severe, sometimes being manifested in high fever, in other cases by a typhoid state. Death is very commonly the sequel (65 per cent.), even if prompt surgical interference takes place. There is generally a history of exposure to infected animals or their hides. Malignant pustule is to be separated from carbuncle by its fulminating character and peculiar appearance.

When an erysipelatoid rash with swelling of the skin and the development of papules, vesicles, pustules, and bullæ appears in association with induration of the skin, with sloughing eventually taking place, the disease may possibly not be erysipelas of a phlegmonous form, but glanders or equina. Numerous inflammatory foci appear in the skin in glanders which end in local abscesses and hemorrhagic nodules, and profound systemic infection is always present. The presence of a sanious discharge from the nose aids in confirming the diagnosis. Death usually comes in a few days in this acute form. Should the course of glanders be chronic, pustules somewhat like those of smallpox, except that they are not umbilicated, lie on an indurated base, and in them is formed a viscid or sanious pus of offensive odor. This disease is rare. Both forms arise from infection from a horse suffering from the malady. Glanders may be confused with variola or the pustular and gummatous stages of syphilis.

The development of pea-sized or larger bullæ upon the skin may indicate the presence of pemphigus, or if there is central nervous disease involving the spinal cord and resulting in trophic lesions similar bullous eruptions may take place. The bullæ, if they contain a dark bloody fluid and are situated upon a limb in which there is an abnormally high temperature, are peculiarly indicative of central nervous lesions, particularly if there is a tendency to dilatation of the capillaries of the skin on slight irritation; but if the temperature of the entire body be raised, the physician should remember that pemphigus is a disease in which there is often marked febrile movement. Sometimes these bulbous manifestations are followed by gangrene in cases of neuritis or other disease causing trophic lesions, such as myelitis and paretic dementia.

Bullæ on the face may follow the ingestion of antipyrine or iodine compounds.

The development of a pemphigus-like eruption in the skin may follow the use of salicylic acid or copaiba.

In the cases of herpes zoster the skin lesion often has its origin in compression of the spinal cord, or in such diseases as tabes, spinal meningeal irritation, and peripheral neuritis.

Glossiness of the skin, in which its minute creases become smoothed out and it appears unduly shiny, often results from chronic disease involving some portion of the nervous system connected with the government of nutrition. Very commonly it results from peripheral neuritis. In addition to shiningness there are often redness and marked thinning or thickening of the cuticle and subcutaneous tissues.

Gangrene of the skin may follow nerve injuries or central nervous lesions. Thus it may follow upon division of a nerve-trunk, or be due to cerebral abscess, in which case the gangrene will be with the other localizing symptoms on the opposite side of the body. The cerebral form develops suddenly and without the prodromal redness of bedsores seen in prolonged illnesses. Similar rapidly developing sloughs and ulcerations of the skin are seen in cases of acute myelitis and in the second and third stages of paretic dementia.

A very interesting condition is the so-called spontaneous gangrene of hysteria. On the skin, generally of the breast of a young girl, a spot develops which feels to her to be hot and burning. The skin soon becomes very white, then in a few hours very red and forms a wheal. This rapidly becomes dark and bluish-black, looking like a burn of sulphuric acid, and a slough finally comes away, leaving a permanent cicatrix.

Gangrene of the skin also follows upon diabetes mellitus, and may involve the scrotum or vulva if the irritation of these parts by the urine is constant. More commonly the toes are affected, and there is this important differential point that in the gangrene of old age with bad vessels the gangrene is usually at the tip of the toe, whereas in diabetic gangrene it is frequently about the ball of the big toe or on the sole or dorsum of the foot. Previous to the development of gangrene there are developed bullæ and other inflammatory changes in the skin which is about to be affected. Kaposi describes a serpiginous form of gangrene affecting the leg in diabetes and a variety of tissue break-down, in which a dermatitis, followed by ulcers

and a lupus-like formation, that also occurs in diabetics. Perforating ulcer of the foot occurs in locomotor ataxia and in paretic dementia.

Closely related, yet quite distinct from angioneurotic œdema, is that condition called Raynaud's disease, symmetrical gangrene, or local asphyxia, according to its severity. The fingers and toes or the nose, with or without exposure to cold, are found to be pale and livid, looking like a hand from which all the blood has been removed by the use of an Esmarch bandage. The part often feels as if "asleep," and is more or less numb and without sensation. To the touch the part is cold and waxy, and it does not bleed when pricked. With the onset of these signs there are often general chilliness and malaise. Often this manifestation speedily disappears, leaving the skin apparently normal; but if it persists, the skin becomes glossy, shrivelled, and looks as if it had been soaked in hot water for hours. When the disease is more severe the pale waxiness is supplanted by cyanosis till the finger-tips, for example, look as if dipped in blue ink; there is often local swelling; the skin is frequently found to be sweating freely and is distended with blood. The skin may rapidly separate from the deeper tissues and become necrotic in patches or *en masse*, and the entire tip of the finger, after becoming black, shrivels up into a condition resembling dry gangrene, which is separated from the sound skin by a sharp line of demarcation. Sometimes small necrotic patches slough out, which leave cicatrices telling of the attack. The prognosis is not bad. The most interesting complication of the disease is paroxysmal hæmoglobinuria.

The development of gangrene of the fingers and toes sometimes follows the prolonged use of bread made from rye which is infected by ergot.

Sometimes gangrene of the skin follows severe attacks of the exanthemata in children who are strumous or very feeble, or who are syphilitic.

Ulcers about the base of the finger-nails should rouse the suspicion of the excessive use of chloral.

Bedsores may develop whenever by long-continued pressure upon any part of the body the local circulation is disturbed, particularly if in addition there is general systemic debility from some exhausting disease, such as typhoid fever. They also develop very speedily, and apparently almost spontaneously, in the course of acute transverse myelitis. Under these circumstances the sacrum is the area most

severely affected. Sometimes these sloughs have been known to develop as early as six hours after the beginning of the attack. Associated with the involvement of the soft tissues the bones may break down, and cellulitis about the rectum and bladder place the patient's life in immediate jeopardy. In hemiplegia, particularly in that which is due to cerebral hemorrhage, bedsores often form on the buttocks, and in paraplegia, from other causes than transverse myelitis, upon the sacrum. They also appear on the heels, inside of the knees, and about the hips in some cases of paraplegia.

Sudden sloughing of the skin of the nates sometimes occurs in cases of intracranial hemorrhages, and is said by Joffroy to be connected with lesion of the occipital lobes.

The value of roseola and rupial eruptions in the diagnosis of syphilis has already been dwelt upon. When the roseola becomes transformed into slightly elevated or bean-shaped spots, irregularly scattered, but sometimes forming groups which are apt to be circular, and if these circles become marginated and then scaly on the edges, resembling lepra, or psoriasis, or even go further than this and develop bullæ and blebs, and when the sores which form are filled with a clear liquid which may become sanious or turbid and on drying leave crusts, the removal of which reveals deeply excavated sloughs, the area of the slough often being as large as a silver dollar, but often irregular in outline, syphilitic rupia is probably the lesion. There is, however, this important differential point, namely, that in specific rupia there is an essential feature, a peripheral ring of induration, whereas in the non-specific form this induration is absent.

If, in addition to these variations, the eruptions are dusky red and leave behind them on healing copperish-looking discoloration of the skin, and appear on areas, such as the flexor surfaces, where ordinary skin eruptions are rarely seen, the diagnosis of syphilis is highly probable. If the eruption is chiefly tuberculated and the turbercles are large and more marked than usual, if they ulcerate and become deep sores, and finally form on healing well-marked cicatrices, tertiary syphilis is to be considered the probable cause.

If, again, we find small nodules under the skin covered by dusky red skin, which finally breaks down and discharges bloody serum, or pus which in burrowing forms discharging sinuses, syphilis of the third stage may again be regarded as a likely cause.

The appearance of hard, dark-brown infiltrated areas in the skin may be due to the excessive use of bromine, and as they gradually

become depressed in the centre closely resemble in some cases the nodules of syphilis.

The skin of the abdominal walls in cases of ascites is apt to be not only thinner than normal but tense and slightly shiny, while its appearance when viewed in a good light may be slightly blue like the iridescence of certain kinds of glassware.

Scars of the skin often give us much useful information. Early tendencies to struma or tuberculosis may be found in the scars resulting from suppurating cervical glands. In the groin such scars may be an evidence of venereal infection, although it should be remembered that suppuration of these glands usually takes place as a result of chancroids and not from true chancre. It has already been shown that syphilitic skin lesions often leave scars to mark their site. Scars upon the head tell us of possible injuries to the brain in suspected traumatic epilepsy, or of falls in epileptics. Similarly, other traumatisms in the history of the patient may be discovered by scars elsewhere.

The presence of numerous regularly arranged fine scars on the chest or elsewhere may develop the fact that the patient has at some time been wet-cupped for some pulmonary or other disease, or if the peculiar three-pointed scar of the leech is seen good evidence of a bleeding is presented.

When the skin of the abdominal wall exhibits stria or scars arranged in parallel series, it indicates that it has been stretched very considerably by pregnancy, ascites, and more rarely by excessive corpulence.

Sweating of the skin, aside from the normal and almost imperceptible exhalation of moisture, takes place in health as a result of severe muscular exertion, whereby the peripheral circulation is increased and the bodily temperature raised, or when the body is very heavily clad or exposed to external heat in excess. In all these cases the sweating is to be regarded as a physiological effort on the part of the body to reduce its temperature by increased evaporation from the surface. In disease sweating provides us with very important information in many conditions.

During the course of fevers, which naturally end by crisis, the occurrence of a profuse sweat (generally associated with a fall of temperature) gives us the first sign of beginning convalescence, and in irritative fevers, or those due to cold and congestion, the artificial production of a sweat is decidedly a good omen. The sweat of crisis

is perhaps most marked in croupous pneumonia. Profuse sweating is also a characteristic symptom of relapsing fever, pyæmia, acute ulcerative endocarditis, phthisis, malarial fever of the distinctly periodic type, and of typhoid fever and collapse. Constant profuse sweating is marked in some cases of acute articular rheumatism, and it is worthy of note that while sweating generally occurs in febrile diseases at a time when the temperature is falling, that in rheumatism the febrile movement may even increase during the sweat rather than decrease.

Profuse so-called colliquative sweats often occur at night in debilitated persons without the presence of any febrile movement, and are but an evidence of profound nervous and vasomotor relaxation. Moderate sweating sometimes is seen from similar causes in feeble persons after taking anything in the food or drink which produces circulatory or nervous excitement. Localized sweatings occur almost solely in subjects of nervous disease, which is often organic, as in paretic dementia, and sometimes functional, as in hysteria or Raynaud's disease. They depend upon perverted vasomotor influences sent to the glands and their supplying vessels in those particular areas. Localized sweating of one side of the face or neck or chest is often a most important sign of a thoracic aneurism pressing on the cervical sympathetic. Bromidrosis may occur in hysteria, or the head may be the only part affected in Graves's disease and in migraine. Profuse sweating of the head of an infant when sleeping may be indicative of rickets.

In cases of the uræmia of cholera or of renal disease the profuse sweating of the skin sometimes results in the deposition of faintly lustrous scales of urea. These are usually seen on the face and nose, commonly on the sides of the nose.

The quality of the sweat varies greatly in many persons. In cases of deficient renal activity it often contains urinary elements, smells uriniferous, and may even deposit particles on the skin in small white scales, particularly on the forehead and nose. This is called uridrosis. In jaundice the sweat may be bile-stained.

Excessive dryness of the skin is seen in grave forms of renal disease, in nearly all acute fevers with a high temperature, and in cholera and diabetes, in which the dryness is largely the result of the drainage of liquids from the body.

Sometimes after a prolonged dryness of the skin during high fever, as soon as sweating begins, hundreds of little blisters develop,

due to retained sweat under the epiderm. These are called miliaria or sudamina.

When the skin is dry and harsh, and the naturally thickened portions have in their folds a peculiar white appearance as if filled with meal, diabetes should be sought for. Rarely the physician may be deceived by profuse sweating in diabetes.

Dropsy and Swelling of the Skin. Swelling of the skin and subcutaneous tissues occurs most frequently as a result of dropsy, in which condition the lymph-spaces become filled by liquid. The skin in the area involved is not only swollen but doughy, or if the effusion is very great the skin may be of almost board-like hardness, so tensely is it distended. Pressure with the tip of the finger upon such an area will result in pitting, and this is one of the more important signs separating dropsy or true œdema from the swelling of acute inflammation, which, while it may be very tense, does not pit. Further, the swelling of inflammation is usually localized, reddened, and feels hot to the touch, whereas the dropsical swelling is more diffuse, is pale, and the temperature of the part is lower than normal. When the effusion of liquid is limited to one portion of the body it is usually called œdema or localized dropsy, whereas if the entire body is boggy it is designated general anasarca. Dropsy is to be separated from myxœdema by the facts that in the latter disease the onset is very slow, the swelling does not pit on pressure, it is universal and fairly equally distributed over the body, the thyroid gland will often be found diseased, the subcutaneous tissues are not boggy but resistant, and there is anæsthesia of the skin. When the subcutaneous tissues are distended by air instead of liquid they are even less resistant than in dropsy, the swelling is usually very localized and does not pit, and the part crackles or crepitates on gentle pressure.

The presence of dropsy is indicative of many widely separated diseases. In the first place, it may indicate a deficient circulation of blood, either by reason of a feeble or diseased heart or because of obstruction by the pressure of growths, thrombi or emboli. It may be due to disease of the walls of bloodvessels and lymphatics, as is generally the case in renal disease, or it may arise from disease of the blood itself. Again, in some cases it is due to disordered nervous control of the vessels, by reason of centric or peripheral changes, which may be organic or functional.

The significance of a widely diffused general dropsy or anasarca

is generally that there is well-marked renal disease, and this probability is greatly strengthened if the œdema of the face be well marked, particularly in the morning on arising, disappearing as the day goes on. The skin in such cases will usually be quite pale, and an examination of the urine will reveal the presence of the signs of nephritis. The next most common cause of general anasarca after renal disease is heart disease. When due to this cause it will be found that the ghastly pallor of renal anasarca is replaced by cyanosis, and often by engorgement of some of the superficial veins, while the physical signs of cardiac disease will confirm the diagnosis. General anasarca may rarely arise as a result of a multiple peripheral neuritis, and it also occurs as a symptom of beri-beri and from the excessive use of large amounts of arsenic. This arsenical anasarca may be due to the neuritis produced by the drug, although Wood thinks it is due to a cellulitis. Rarely we find general anasarca in cases of advanced cancerous cachexia, and care must be exercised that the hæmic murmur due to anæmia does not mislead the physician into a diagnosis of heart disease.

Dropsy widely diffused or localized in the feet and legs also occurs in scurvy.

The most common seat of localized dropsy or œdema is the feet and legs, particularly about the instep, the ankles, and the tibiæ. When it is bilateral it is generally indicative of cardiac failure or more rarely of renal disease. Nearly always, if it be renal, a careful examination will discover the œdema in other parts of the body, although it may be most marked in the feet and legs. In many cases the various serous sacs, such as the pericardium, peritoneum, and pleuræ, will be found to contain more liquid than normal, and the tissues generally will be found infiltrated.

Other causes of œdema of the feet and legs are anæmia, and obstruction to the return of flow from the lower limbs by reason of growths in the abdomen pressing upon the iliac veins or inferior vena cava. Thus cancer of the pancreas sometimes causes œdema of the feet and legs in this manner. Very rarely œdema of the lower extremities follows hepatitis or cirrhosis of the liver as a primary symptom. Usually such lesions produce ascites alone, or if the legs are involved they become so by reason of the pressure of the fluid in the pelvis, during the time that the patient is sitting up or standing. This latter cause of bilateral œdema of the lower limbs is, however, rare. Sometimes œdema of both legs and feet comes on in persons,

who, though feeble and relaxed, remain standing with little muscular movement during many hours in the pursuit of their occupation, as in typesetters and salesmen. In other instances, very much more frequently, œdema of the feet and legs comes on in the course of profound anæmia, resulting from slow hemorrhages or other causes. It is also seen in the cachectic stage of gastric cancer, owing to the anæmia which is present.

When the face is œdematous the swelling is most marked under the eyes, the lower lids of which are particularly puffy in the morning and nearly normal in appearance at night. This form of œdema is most marked in, and almost pathognomonic of, renal disease. Its only other cause is the excessive taking of arsenic, and angioneurotic œdema. More alarm should be felt at a slight swelling of the face of this character than if the feet are markedly puffed. Sometimes œdematous swelling of the side of the face and scalp which has been involved in a severe attack of neuralgia, takes place.

When œdema of one or both eyelids occurs, with protrusion of the eyeball, the swelling extending to the rest of the face as time goes on, it forms an important symptom in obscure cases of suspected cerebral thrombosis, and is caused by the intimate association between the intracranial vessels and those of the face.

Sometimes œdema of the eyelids comes on in neurotic subjects and may extend to the forehead. This may be seen in children, most commonly about puberty, and is probably a neurosis.

Œdema of the upper extremities alone only results from causes interfering with the flow of blood, such as is produced by morbid growths in the chest, as mediastinal growths, and in cases of aneurism. When the swelling is limited to one arm or leg it is a sign that there is interference with the local circulation, as, for example, the obstruction of the femoral vein by thrombus, as in phlegmasia alba dolens, following labor or enteric fever, or, when the œdema is in the left leg, by cancer of the sigmoid flexure. If the swelling of the arms and head is manifested suddenly, it is probably due to that rare condition in which an aortic aneurism ruptures into the vena cava; whereas if it develops slowly, it is due to pressure by a growth.

There remain three forms of local œdema of some diagnostic significance, namely, that occurring in a limited area over some deep-seated suppurative process, as in the skin back of the ear in cases of mastoid abscess or thrombosis of the lateral sinuses, and over the ribs in cases of purulent exudation into the pleura, or on the thigh

in the deep muscular abscesses which sometimes follow typhoid fever.

Œdema of the legs and wrists sometimes complicates relapsing fever and is evidence of profound feebleness, without necessarily indicating renal or cardiac disease. Such a limited œdema, or even general anasarca, may occur during convalescence from typhoid fever from similar causes. Unless this effusion is associated with signs of grave renal or cardiac mischief the prognosis, according to Lendel, is favorable.

When the skin is pale and affected by an œdematoid swelling, with thickening, hardening, and loss of elasticity, particularly about the face, and also in the trunk and extremities, and if this swelling, which resembles œdema, fails to pit on pressure, the physician should remember that myxœdema, or the cretinoid œdema of Gull, may be present. If in addition to these signs there is a half-idiotic or heavy expression of the face, a slow and clumsy manner of speech, with thickened, clumsy fingers, the diagnosis is made practically certain. The brain in this disease perceives or grasps ideas very slowly, and all the functions of the body seem torpid.

There are several other diseases in which great thickening of the skin takes place, which cannot, however, be confounded with myxœdema. In elephantiasis there is a hypertrophy of skin and subcutaneous tissues, which is confined to some particular region of the body and arises from local circulatory disturbance in the blood and lymph-vessels. The skin is very hard, so that the leg, if affected, feels like a solid mass of wood. The disease most commonly affects one of the legs, rarely both, and the scrotum. In both myxœdema and elephantiasis the disease develops very slowly.

When the skin is dotted with irregular patches or streaks, which may be depressed, elevated or tightly stretched, or if the entire skin is thickened, covered with thin scales, or possesses a plaster-like appearance, the physician should recognize these symptoms as indicative of scleroderma. If in addition to these signs there is a fleeting pitting of the skin on pressure and it cannot be pinched into a fold, the diagnosis is confirmed. Sometimes the skin in scleroderma seems bound down by tense cords or streaks of retracted connective tissue.

If during the first months of life the skin of an infant becomes œdematous, hard, tense, and glossy, varying in color from a white to a reddish or dirty yellowish-brown, and if this rapidly involves the entire surface so that the integument becomes cool, immovable,

and resistant, so that the child appears as if frozen into stiffness, it is probably suffering from sclerema neonatorum, a disease entirely different from the scleroderma of the adult. As a rule, death speedily ensues, but before this takes place the parts first affected become thin and lose their swelling and may develop cyanosis and gangrene.

The affection just described is to be separated from œdema neonatorum, a condition arising in prematurely born children. Within a few days after birth there is discovered a pallid, cold condition of the buttocks, thighs, legs, and arms. These parts speedily become œdematous and livid blue. Finally the œdema may become very marked and the skin tense in consequence. Intense drowsiness is a characteristic of the disease. Death commonly ensues, but recovery may occur. While the color of the skin may be identical in œdema neonatorum and sclerema neonatorum, the former affection lacks the stiffness of the jaws and other joints, and the pitting on pressure is marked. As scleroderma does not occur before the first year it can be excluded from the diagnosis.

Very closely allied in its causes and appearance with urticaria of the severe type is angioneurotic œdema. In this condition there appears upon the skin numerous patches or plaques of circumscribed puffy swellings, which have a red appearance and vary from the size of a nickel to a silver dollar or larger. There is an absence of itching, an important difference from true urticaria, but the part affected may be tense or hot to the patient. These attacks are generally recurring, and take place in neurotic persons. They may cause loss of sight through swelling of the eyelids and, where the mucous membranes of the pharynx and larynx are involved, serious interference with breathing. The swelling of angioneurotic œdema does not pit, and it is to be separated from the blue œdema and the white œdema of hysteria. True angioneurotic œdema is rare in hysteria, and if localized swellings do result from this condition the physician will generally find marked hysterical signs of the kind manifested, such as disorders of sensation or tenderness over the ovaries.

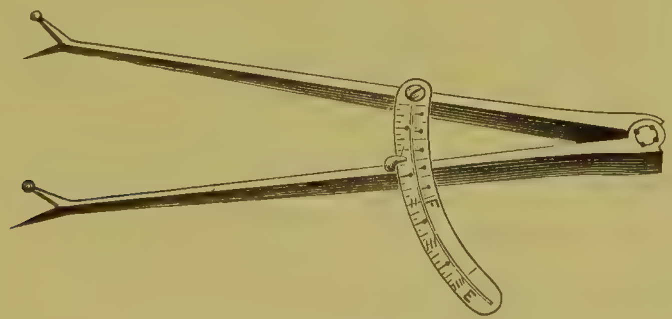
The ocular appearance and touch of the skin having been studied in so far as its surface affords evidence of more deeply seated disease or functional disturbance, we next pass to a study of its sensibility, having the same diagnostic objects in view.

Sensation in the Skin. Before considering the various perversions of its sense it is important to remember that the sensibility of the skin may be divided into four parts, namely, its tactile sense, its

pain sense, its thermic sense, and its sense of pressure. Any one of these senses may be perverted or in abeyance without the others being affected in a similar manner, and it is noteworthy that while corresponding areas of the skin in all individuals have practically identical sensibilities, each part of the skin has a sensitiveness of its own, so that while in some parts the slightest touch is felt, in others severe irritation must be produced to cause much of a result. These differences have been carefully studied by many observers, the most thorough being Weber, who has found that the average tactile ability to separate points brought in contact with the skin is about as follows: At the finger-tips points can be separated at from 2 to 3 millimetres, on the lips 4 to 5 millimetres, on the tip of the nose 6 millimetres, on the cheeks and backs of fingers 12 millimetres, and on the forehead 22 millimetres. The skin on the neck separates points at 34 millimetres; that on the forearm, on the lower leg, and back of foot at 40 millimetres, on the chest at 45 millimetres, on the back at 60 millimetres, and on the arm and thigh at 75 millimetres. If tests be frequently repeated in a single individual the ability to separate the points increases with training. Care should always be taken that the pressure on both points is equal, applied simultaneously, and that the points are equally sharp.

In testing tactile sensibility, not only should points be used but also objects. Often single points may be applied without any abnormal manifestation, and in some cases of disease the skin, which seems devoid of sense on ordinary touch, is found to be excessively hyperæsthetic if the hand is drawn lightly over it.

FIG. 90.



Carroll's æsthesiometer.

The best apparatus for testing tactile sensibility is the æsthesiometer of Carroll, which is a pair of compasses connected by a graduated scale. (Fig. 90.)

The ability to distinguish pain-giving and thermal applications is most acute in the normal skin of the hands, in which tactile sense is also most acute. The methods by which we test the pain sense are several, but chiefly by pricking the skin, more or less deeply, by some sharp-pointed instrument, such as a pin, or by pinching the integument. The thermal sense is also studied by applying bodies which are hot or cold against the skin, such as a cold knife, a small piece of ice, or a test-tube which contains very cold or hot water.

In all such tests the physician should use both hands simultaneously. With one he should apply his instrument to the suspected area, and with the other a similar instrument to a known to be healthy area, in order that an actual comparison as to the sensations may be noted by the patient. Thus the face may be used as the normal area in a spinal lesion, and the skin of the arms as a control surface in a lesion involving the legs. The eyes of the patient should be blindfolded, and if tactile sense is being tested the instrument must be of the same temperature as the body.

Disturbances in the sensation of the skin may arise from functional or organic disease, involving the peripheral nerves, the sensory tracts in the spinal cord, similar tracts in the lower part of the brain, and, finally, the subcortical or cortical parts of the cerebrum itself.

FIG. 91.

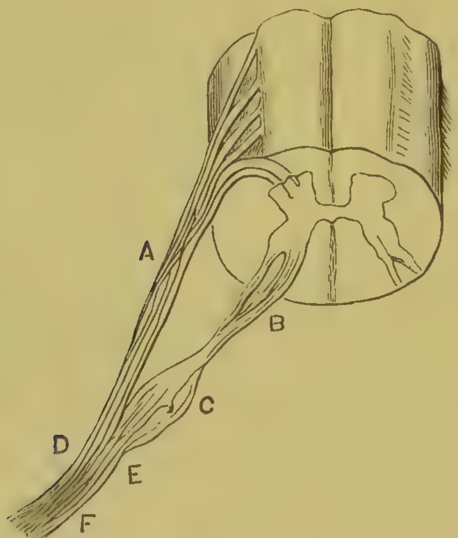


Diagram of the relations of the nerve-roots to the spinal cord. A, Anterior root; B, posterior root; D, motor part, E, sensory part; F, mixed nerve. (EDINGER.)

The sensory pathway or the afferent fibres pass upward, starting with the peripheral sense organ in the skin, or elsewhere, and after

forming part of the nerve-trunk and entering the ganglion on the posterior root, enter the spinal cord by what is called the posterior root, which is shown in the figure near the words "Lissauer's zone." (See Figs. 91 and 92.)

FIG. 92.

Chart showing the sensory tracts in the spinal cord with entrance of the sensory nerve-roots.

The posterior root enters the cord in three sets of fibres; one of these, the one lying nearest the posterior median fissure, is composed of coarse fibres and is called the median bundle, and passes obliquely into the lateral part of the column of Burdach.

Some of these fibres also enter the column of Goll. As soon as they have entered this column they turn at right angles and run upward for some distance, thereby helping to form the column of Burdach. Some of them also run downward a short distance.

The second set, nearer the side of the cord, goes directly into the gray matter of the posterior horn through the substance of Rolando, and the third set, nearest the side of the cord, enters the cord very superficially, and, turning at once at a right angle, goes upward to form Lissauer's zone. The longitudinal course of these fibres is shown in Plate VI. Here it is seen that they pass upward chiefly in the column of Goll (posterior median) to the medulla oblongata. Before reaching the medulla, however, the column of Goll ends in the gracile nucleus and the column of Burdach in the cuneate nucleus.

These nuclei which have received the fibres of the two sensory columns give origin to fibres which pass to the brain. They sweep forward to the front of the central canal of the medulla and decussate at a higher level than the motor tracts. A great majority of these fibres pass upward to the brain, but some pass forward, and finally join the restiform body on the posterior aspect of the medulla. Those which pass upward from the so-called fillet pass into the crus cerebri, in that part of it called the tegmentum, and from thence into the posterior part of the posterior limb of the internal capsule, from whence they spread out in the corona radiata to the occipital lobe and temporo-sphenoidal lobes.

The duty of the physician in all cases is to determine first whether the disease is functional or organic, and then where the lesion producing the symptoms is situated.

The two chief manifestations of perverted sensibility in the skin are anæsthesia and hyperæthesia, and the minor ones are paræsthesia or numbness, tingling and formication, and analgesia, or the failure to feel pain. Whatever the cause of these symptoms may be the history of the patient and his general symptoms should be carefully studied when examining these signs, as frequently a diagnosis with them alone as guides is imposible.

Anæsthesia. Anæsthesia of the skin is indicative of a very large number of conditions, arising anywhere in the sensory appa-

PLATE VI.

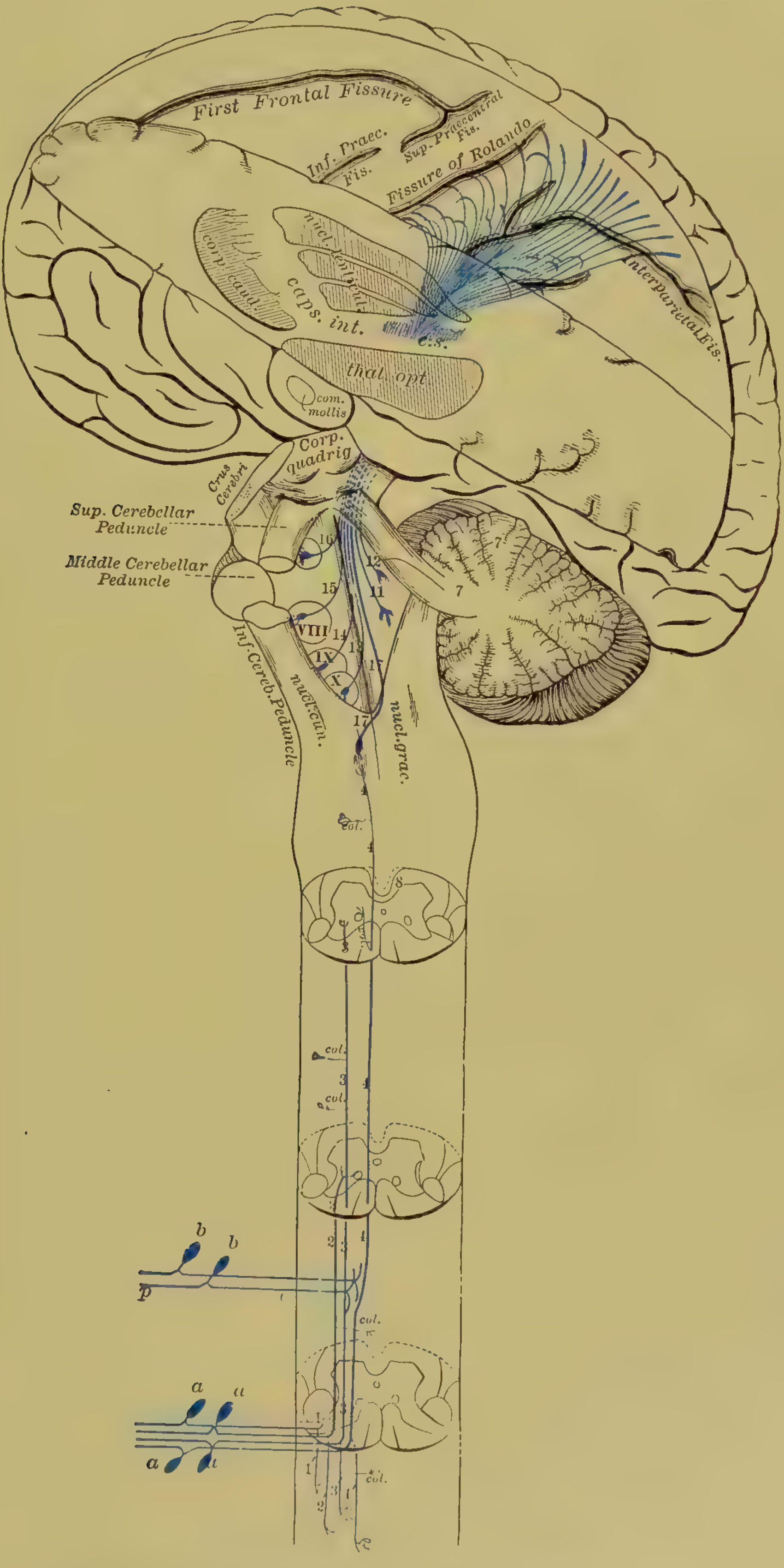


Diagram showing Course of Sensory Fibres from Periphery to Cord, Cerebrum and Cerebellum. (Flatau.)

ratus. In other words, anything which interferes with the transmission of an impulse to the perceptive centres in the brain may be its cause. Of the functional causes, the most frequent is hysteria, and the presence of anæsthesia in a female should always arouse a suspicion of its being due to this cause. Rarely it is seen in hysterical males. The organic causes of anæsthesia of the skin are cerebral hemorrhage, cerebral tumor, hemorrhage of the pons or tumor of the pons, hemorrhage of the cord, tumor of the cord, myelitis (transverse), locomotor ataxia, cerebro-spinal meningitis, spinal meningitis, compression of the cord by vertebral caries, by fractures, by dislocations, and hemorrhage into its membranes. Additional causes are pressure on the posterior nerve-roots, by reason of caries and growths, inflammation of the nerves (neuritis), injuries to the nerves by blows, pressure, or cutting, and, finally, by paralysis of the nerve-endings by cold or the action of drugs.

Anæsthesia, because of its area of distribution, may be divided into hemianæsthesia, crossed anæsthesia, bilateral anæsthesia, irregular but complete anæsthesia, and partial anæsthesia.

HEMIANÆSTHESIA occurs most frequently as a result of hysteria, next most commonly from lesion of the posterior part of the internal capsule, and more rarely from spinal injuries or growths in the cord of a unilateral character.

The hemianæsthesia of hysteria involves, as its name implies, one side of the body, and is usually universal on that side, except that here and there there may be patches of hyperæsthesia or tenderness, dotted like oases in the midst of the absence of sensation. This anæsthesia is often unaccompanied by motor paralysis, and its area is separated from the opposite side of the body by a sharp line of demarcation, which runs along the middle of the trunk and face. The presence of such a well-defined line of separation in a young woman is of great significance of hysteria. The anæsthesia is generally absolute, and severe injury may be done to the skin in some cases without the patient feeling it; but, notwithstanding its intensity, it is a noteworthy fact that the anæsthesia may transfer itself to the opposite side of the body with great suddenness, and equally suddenly return to its early site. In a great majority of cases, for some unexplained reason, the left side is the one affected by anæsthesia, and hyperæsthesia of the opposite side increases the contrast which exists between it and that in which sensation is lost. (See Hyperæsthesia.) In some cases of hysterical hemianæsthesia

the paralysis of sensation involves the nerves of special sense, and loss of smell, taste, hearing, and impairment of sight may ensue on the same side. The peculiarity of the visual changes are so characteristic that they practically decide the character of the case when they are discovered in any instance of doubtful diagnosis, and consist in a loss of the color-vision (first, violet is lost, then blue, and then red) and a great limitation of the visual field, whereas in the hemianæsthesia due to an organic lesion in the internal capsule, so situated as to involve the nerve-fibres connected with vision, there is hemiopia. Hemianopsia, due to hysteria, is so rare as to be denied by most authorities, but Lloyd and de Schweinitz have seen a case. Generally the loss of vision on the anæsthetic side is a total one for both sides of the eye in hysterical blindness. (See chapter on Eye.) Nearly always in hysterical hemianæsthesia a spot can be found over the shoulder which is not anæsthetic. The age of the patient, her sex, the general expression of the face, and the history of her illness, associated, as is frequently the case, with some, or all, of the hysterical symptoms detailed further on in this chapter, will generally decide the diagnosis in favor of hysteria.

A form of hysterical hemianæsthesia very apt to lead to an error in diagnosis is that seen in persons who have suffered from infantile cerebral paralysis with the resulting deformity (a disease not characterized with sensory disturbances), but who have in later life superimposed upon the old picture of disease that of hysteria with this sensory manifestation.

Anæsthesia, irregular in its distribution, or absolute hemianæsthesia, may occur in the course of chorea. The presence of chorea in its motor manifestations clears up the diagnosis as to the cause of the loss of sensation.

Hemianæsthesia when not hysterical is nearly always due to an organic lesion in the posterior part of the hinder limb of the internal capsule on the opposite side of the brain from the anæsthesia, and the additional symptoms which sometimes accompany it depend for their existence upon whether the lesion is large enough to involve, not only the fibres from the cutaneous areas, but also those of special sense, such as sight, hearing, or taste. Nearly always the area destroyed is sufficiently large to result not only in hemianæsthesia, but also loss of motion on the same side. The loss of sensation in such a case is rarely as complete as in hysteria, and the sole of the foot and palm of the hand are often not affected. In rare instances,

however, the hemianæsthesia of capsular disease may be absolute and universal, or, more rarely still, occur in patches, thereby closely resembling the anæsthetic patches seen in hysteria.

Hemianæsthesia may also be produced by a large lesion of the cortex in the occipital, temporal, and parietal lobes, in which case it will involve the side of the head as well as the trunk, and will be associated with such definite evidences of apoplexy or injury that the diagnosis will be readily made. If it is widespread, all the special senses will be involved.

Sensory disturbances of the skin are more frequent in softening of the brain than in hemorrhage into the brain, and most commonly are associated with subcortical, rather than cortical lesions.

In this connection it should be remembered that the irregularity of distribution of the lesions in disseminated sclerosis may cause a hemianæsthesia, partial or complete.

Anæsthesia resulting from tumor of the brain occurs in about 20 per cent. of the cases of brain-tumor, and may be unilateral and confined to the paralyzed side, or appear as an isolated symptom without motor paralysis. When of the latter form it is often associated with lesions in the neighborhood of the fissure of Rolando, and in tumors involving the posterior parietal region and the posterior part of the internal capsule.

Autopsies and experiments show that hemianæsthesia may arise from a lesion in the optic thalamus, but such an occurrence is very rare.

A very important and essential factor in making the diagnosis that the anæsthesia is cerebral in origin is the history of the beginning of the attack, which has been sudden if due to hemorrhage, embolus, or thrombus (see Hemiplegia), and characteristic of the condition which we call apoplexy.

Unilateral anæsthesia associated with motor paralysis, both being somewhat irregular in their distribution, may be due to a lesion, such as a tumor in the pons or medulla oblongata, but death so commonly ensues soon after the apoplexy that the symptom is often overlooked or cannot be developed. Further, the discovery of such anæsthesia does not positively localize the lesion in the pons, for we do not know much about the course of the sensory fibres in this part. If, however, the area supplied by the trifacial nerve, namely, the face, is anæsthetic, and these symptoms are associated with it, then it is fair to assume that the trouble lies in the pons and

has involved the nucleus of the fifth nerve. (See Anæsthesia of Face.)

Anæsthesia of irregular distribution or confined to one limb may result from cerebral or spinal lesions, or be due to a neuritis, of which form we shall speak further on. If it is a monoanæsthesia from cerebral disease, which is very rare, the anæsthesia is most marked at the distal part, and gradually fades off as the trunk is approached. It is evenly distributed, so far as circumference is concerned, and has no sharp line of demarcation.

When such an anæsthesia is due to spinal disease the cause may be tumor of the spinal cord, the symptoms depending in their character on the area involved; but in any event the upper border of the area involved is sharply outlined and a constriction-band sensation is often present.

The irregular form, due to hysteria, has the same general peculiarities of migration as are seen in hemianæsthesia from this cause, and in its symmetrical form it closely resembles the anæsthesia due to multiple neuritis. Thus, in the hand the area of anæsthesia may be that covered by a gauntlet glove, in the foot that covered by a sock, the line of normal sensation being present just above the place to which these protections usually extend.

Crossed Anæsthesia. When sensory paralysis of one side, associated with partial paralysis of motion or paresis on the same side, comes on, and with it there is hyperæmia of the skin on that side from vasomotor paralysis, there is a strong probability that there is a lesion in the cerebral peduncle of the opposite side. If there is at the same time paralysis of the muscles supplied by the oculomotor nerve on the opposite side from the anæsthesia—that is, on the same side as the lesion, this diagnosis is still further confirmed; and if the tongue and half of the face of the paralyzed side of the body are paralyzed, still further confirmatory evidence of a peduncular lesion is obtained. Thus there might be hemianæsthesia and paralysis of the right side of the body, including the face and right half of tongue, and ptosis, from oculomotor palsy, on the left side of the face. The paralysis of the body, face, and tongue would be on the side opposite to the lesion, but the oculomotor paralysis would be on the same side as the lesion.

Crossed anæsthesia of the limbs and face—that is, anæsthesia of one side of the body with anæsthesia of the opposite side of the face—can only occur in lesions involving the upper part of the pons in

such a way that the fibres of the trifacial are diseased on one side, and the path for sensory impulses of the other side of the body is also destroyed. (See chapter on Face, Head, and Hemiplegia.)

An important point to be noted in the diagnosis of cerebral anæsthesia is the fact that the reflexes are preserved, though the patient may not feel the touch or painful impression; that is to say, irritation of the skin causes movement in the arm or leg, not by any intention of the patient, but owing to the fact that the sensory centres in the cord receiving an impulse cause the corresponding motor centres to send out impulses which contract the muscles.

Partial hemianæsthesia, with partial hemiplegia on the opposite side in crossed paralysis, may occur from lesions on one side of the spinal cord, and, if high up, involve a large part of the trunk and lower limbs. (See chapter on Feet and Legs, part on Myelitis.) These cases have been explained by a theory of Brown-Séquard, which has recently been doubted owing to the studies of Mott and others. Thus, until recently, it has been considered as proved that sensory impulses entering the cord crossed to the opposite side almost at once, at least in greater part, passing to the lateral columns in front of the pyramidal tract, and that a very small number entered the posterior columns, while a few ascended in the gray matter. The recent studies of Mott, in confirmation and criticism of still other investigators, seem to prove that the reverse is the case and that the greater part of the sensory impulses do not cross the cord, only a few fibres passing to the opposite side on entrance. He believes that the main pathway for heat and cold sensations is in the gray matter, while the tactile pathways are in the posterior columns, although it is possible that some few isolated fibres may exist in the lateral columns and that these cross in the cord about the level of entrance.

Bilateral Anæsthesia. Anæsthesia of hysterical origin involving both legs, and sometimes the lower part of the trunk on both sides, may occur, and, aside from the typical signs of hysteria in general which distinguish it, may be discovered by the fact that in hysteria the failure of sensation does not involve the skin of the genitals, as it does in organic lesions producing somewhat similar symptoms. In addition it will be found that in hysteria a V-shaped piece of skin over the sacrum is not anæsthetic. Anæsthesia of this variety, corresponding in the sensory organs to what we call paraplegia in the motor apparatus, is practically never produced by a cerebral lesion,

and, if not hysterical in cause, must be spinal; but it is much more rare than is motor paralysis in these parts from lesions in the spine. When it does ensue from spinal causes motor paralysis will in the great majority of cases be found associated with it at least to some extent. To express it concisely, the characteristic of a typical spinal anæsthesia is that it is bilateral and usually involves both sides quite symmetrically; that motor paralysis is generally associated with it; that the reflexes are greatly perverted; and that trophic changes may be present as a result of an involvement of the trophic cells in the anterior cornua coincidently with the disease of the sensory parts of the cord.

The diseased conditions of the cord which result in symmetrical anæsthesia of the skin of the legs and trunk are, first and most prominent, locomotor ataxia; second, myelitis; and, finally, hemorrhages, tumor of the cord or its membranes, meningitis, or injuries which cause pressure on the sensory tracts by producing fracture of the vertebræ or dislocation. Very rarely, however, a lesion of the pons may so result.

Anæsthesia of the lower portions of the body and legs occurs in the later stages of locomotor ataxia, and is usually preceded by forms of paræsthesia. (See Paræsthesia.) The anæsthetic areas are most marked in the soles of the feet and about the malleoli, according to Belmont. In other words, blunting of sensibility is seen in nearly all cases of tabes dorsalis late in the disease. In some cases the sense of touch is preserved and the sense of pain lost (analgesia), while in others the opposite condition is present. Again, we find loss of tactile sense and of pain-sense without loss of heat and cold sense and *vice versa*. A very characteristic sensory symptom of tabes is the delay in the recognition of an irritation of the sensory nerves, so that if the patient be blindfolded and then pricked with a pin he will not make an exclamation or draw his foot away for several seconds. In other instances the patient complains of repeated pricks when only one has been given, or, when asked the number of points pricking him, states that there are four or five instead of the one really present. If, in addition to these sensory disturbances, we find Romberg's symptom (see Legs), Argyll-Robertson pupils (see Eye), and loss of patellar reflex (see Reflexes), and a number of the other diagnostic peculiarities of tabes, the decision as to the cause of the anæsthesia is easily made.

Slight anæsthesia, retardation of the transmission of sensory im-

pulses from the skin, and perversions of temperature-sense may be rarely developed late in the course of Friedreich's ataxia.

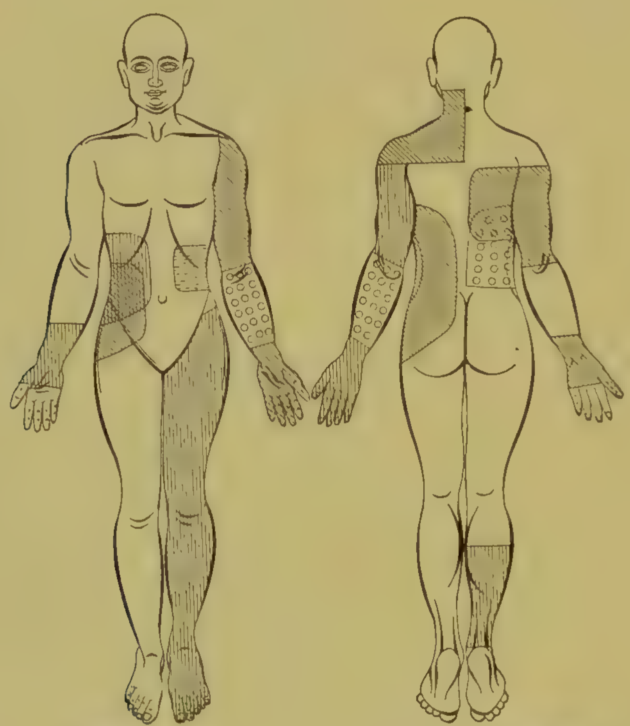
Bilateral anæsthesia of the character just discussed, as caused by locomotor ataxia, may also occur as a result of acute or chronic myelitis. The first change under these circumstances is a mere obtunding of sensitiveness, which gradually deepens till loss of pain-sense, pressure-sense, and, lastly, complete anæsthesia is developed. The development of these symptoms indicates involvement of the posterior columns. Loss of reflex activity in the legs is developed in direct proportion to the destruction of the motor and sensory nerve-tracts in the cord. The predominance of motor paralysis, the fact that the lower limbs are both involved, and the absence of the characteristic symptoms of locomotor ataxia all tend to make the diagnosis certain, while the absence of the pains of tabes and the other signs of that disease still further exclude its presence from the case. Further than this, the myelitis creeps up the cord, involving new areas, and new parts of the skin become anæsthetic. An important point, too, in regard to the anæsthesia of acute myelitis is this, namely, that while in the upper extremities the loss of sensation and motion is associated, so that both functions are lost in the same area, in the lower extremities these two functions are not lost in the same areas. Thus myelitis of the lumbar enlargement in its lower part is accompanied by anæsthesia of the gluteal area and motor paralysis of the anal muscles; and, again, anæsthesia of the gluteal region, the back of the thigh, and the back of the calf is associated with loss of power in the muscles that move the foot, while in lesions of the upper part of the lumbar segment the anæsthesia involves the thigh, the inner side of the leg and the foot, in association with paralysis in the quadriceps extensor and deeper muscles of the thigh. (See chapter on Feet and Legs, part on Myelitis.)

The development of sudden bilateral anæsthesia, which is accompanied by severe pains of a tearing or burning character, creeping rapidly up the body, is indicative of acute hemorrhage into the spinal membranes, or it may be due to that very rare lesion, hemorrhage into the cord. In either case motor paralysis is present. Anæsthesia, or the milder perversions of normal sensibility of the skin, may be present in cases of compression of the cord by caries, and by curvature, tumors, or aneurisms producing erosion. Sometimes, while tactile anæsthesia is complete in these cases, severe pain is con-

stantly suffered (anæsthesia dolorosa), and this is often the case, according to Wood, in cancer of the spine.

Partial anæsthesia of the skin of the trunk and arms of a bilateral character, associated with progressive muscular atrophy, scoliosis, and trophic lesions in the skin, points strongly to syringomyelia. The loss of pain and temperature-sense is usually the first symptom. The areas of anæsthesia are best shown in Fig. 93.

FIG. 93.



Sensory chart, showing areas of . . . . . *Thermo-Anaesthesia*, *Analgesia*, *Thermo-Anaesthesia, and Analgesia*, *Tactile Anaesthesia*, and areas in which the patients answer to tests of temperature showed reversal *Cold-Hot; Hot-Cold.* (DERCUM.)

Having considered the general spinal causes of anæsthesia of the skin, it yet remains to determine what part of the cord is involved by the pathological process; and this is fortunately possible, chiefly through the very accurate and noteworthy studies of M. Allen Starr, Thorburn, and Head, not to mention collateral ones of great value by Horsley and many others; but the field is only partly covered, and some of our uncertainties depend upon lack of knowledge as to the course of the sensory fibres in the cord.

Roughly, we may state that disease of the cervical cord generally produces disturbances of sensation in the arms, hands, and fingers;

disease of the dorsal cord, disturbances in the sensation of the back and trunk, which may radiate into the thighs; and disease of the lumbar cord gives rise to these symptoms in legs and feet.

FIG. 94.

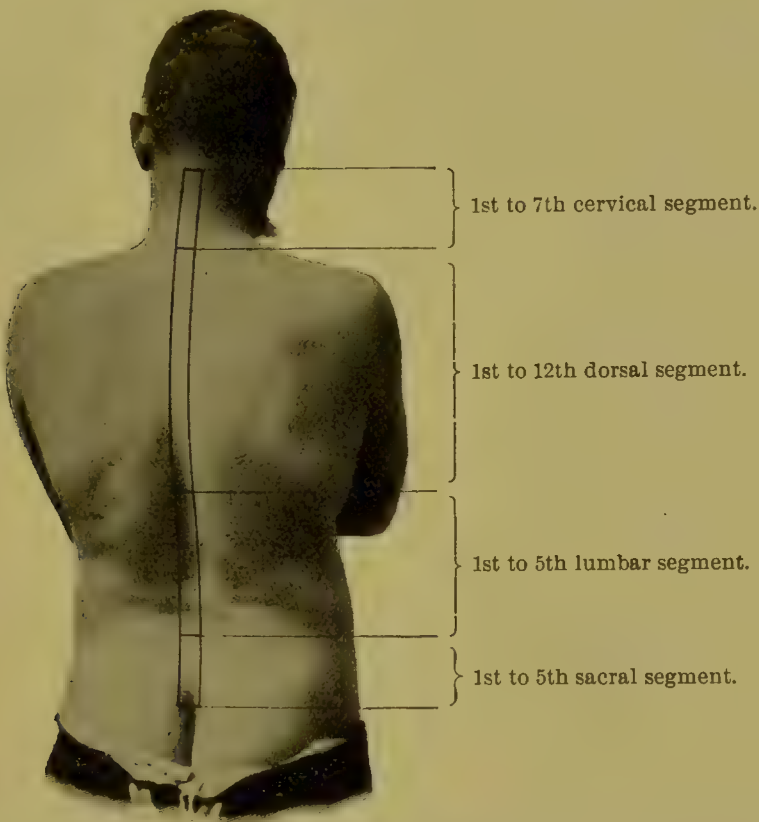


Diagram showing the surface-areas of the back corresponding approximately to the areas of the spinal cord supplying the trunk and limbs.

Again, it is to be remembered that, as a rule, in a transverse lesion of the spinal cord the anæsthesia begins at a level which is three or four inches below the lesion in the cord (Horsley and Gowers); this being due, as proved by Sherrington, to the fact that each area of skin is supplied by three nerve-roots whose peripheral filaments overlap one another.

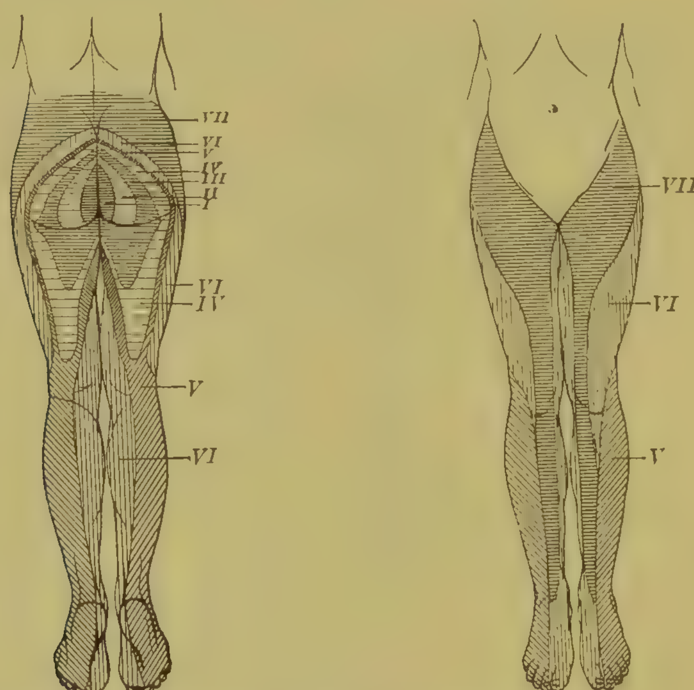
For the ready study of the subject the cord has been separated into segments corresponding with the vertebræ covering it.

The areas of anæsthesia produced by spinal injury or disease are best described in Starr's well-known article and diagrams, from which we quote. In this connection the reader should refer to the table on pages 97 and 98, showing the localization of the functions of the segments of the spinal cord. (See chapter on Legs and Feet.)

The anæsthetic areas included in zones I. and II. in Fig. 95 are

due to a lesion involving the conus medullaris and the fourth and fifth sacral segments of the cord. These zones include the perineum, the posterior part of the scrotum in males, the vagina in females, and the mucous membrane of the rectum. Anæsthesia in zone III. is due to lesion of the third, fourth, and fifth sacral segments, and includes a large part of the buttock and the upper part of the thigh, posteriorly, in a triangular space. Zone IV. is practically an enlargement of zone III. in every direction, particularly toward the popliteal spaces, and is probably due to a lesion in the first and second sacral seg-

FIG. 95.



Areas of anæsthesia in lesions at various levels of the spinal cord from sacral V. to lumbar II. (After STARR.)

I. Sacral V.
II. Sacral IV.
III. Sacral III.
IV. Sacral I.
V. Lumbar V.
VI. Lumbar III.
VII. Lumbar II.

ments; but this needs confirmation, as Starr points out, by autopsy. Zone V. includes all the first four zones just named, and extends down through the popliteal space in a band-like shape; after it passes this space it descends the outer side of the leg and foot, sometimes ending at the ankle, sometimes at the sole or the three outer toes and half the next toe. Such an area indicates a lesion involving all the segments of the sacral cord, and extending into the lumbar cord to the fifth lumbar segment. Zone VI. is caused by a lesion extending

to the third lumbar segment, and when it is present the anæsthesia covers the back of the thighs and legs and also the front of the thighs, except in an area which extends from above downward along the shin, sometimes to the foot, as in Fig. 95. If the foot is involved, the lesion in the lumbar cord is probably above the third lumbar segment. Zone VII., which is larger than all, follows a lesion in of the four lumbar segments—that is, all but the first. The line of anæsthesia, Starr tells us, is lower in front than behind. When the abdominal wall is involved in the anæsthesia the first lumbar segment is probably diseased.

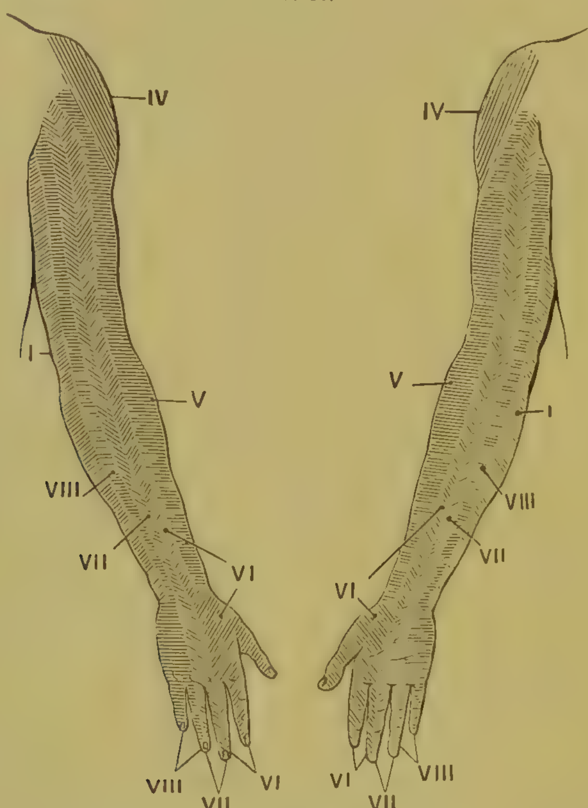
The ascent of anæsthesia from the abdomen up corresponds very closely to the levels in the cord if we allow for the space already mentioned, of two to three inches for the interlacing anastomosis of the nerve-fibres of the posterior roots.

They are about as follows, according to Thorburn : When the anæsthesia is as high as the anterior inferior spine of the ilium, the lesion is at the twelfth dorsal vertebra ; if at the umbilicus, at the eleventh and twelfth dorsal vertebra ; if up to the lowest floating rib, the whole eleventh dorsal vertebra ; if to one to four inches above the umbilicus, the ninth and tenth dorsal, and perhaps part of the eighth dorsal vertebra ; if as high as the nipples, the fourth dorsal vertebra ; and if to the third rib, the lesion is as high as the second dorsal verterba.

Starr has also given us in another paper than that already quoted equally good ideas of the areas of anæsthesia occurring above those just described. (Fig. 96.) When the anæsthesia extends to the arms and is found upon the inner side of the arm and the forearm, reaching to the wrist, but not to the hand, and also involves a small zone on the extensor and flexor surface of the arm and forearm, the second dorsal region is the site of the lesion. If the anæsthetic area includes the ulnar side of the hand, the palmar and dorsal surfaces of the same, and the little ring-finger, and extends in a narrow strip up to the axilla on both the anterior and posterior surfaces of the arm and forearm, the lesion is probably at the level of the eighth cervical vertebra. When the zone involved extends to the middle of the central finger on the palmar and dorsal aspect, and runs up the centre of the forearm and arm, the seventh cervical area is diseased. Again, when the remaining skin of the hand up to the wrist and a narrow strip of skin up the forearm and arm on both surfaces to the axilla is affected, the lesion is at the sixth cervical vertebra, while

anæsthesia of the forearm and arm on the outer surface as high as the deltoid insertion indicates that the fifth cervical vertebral area is in trouble. Lesions higher than this usually produce death before it is possible to test sensibility.

FIG. 96.

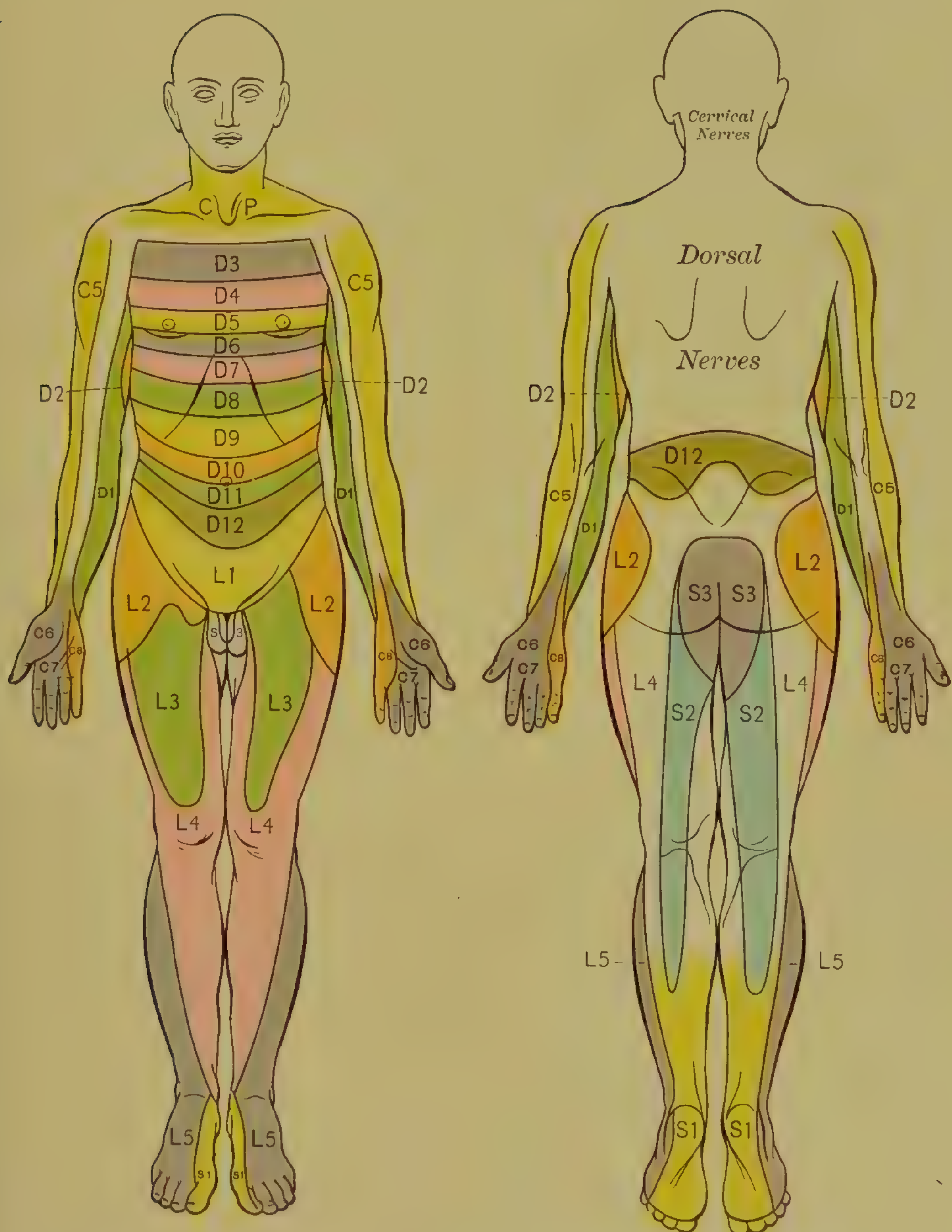


Areas of anæsthesia from lesions and various levels of the spinal cord from the second dorsal to the fifth cervical. (After STARR.)

In order that the reader may gain a still better idea of the probable, or rather approximate, area of distribution of the spinal nerves, the three charts of Thorburn, Starr, and Head are here reproduced, as prepared in colors, with significant lettering by Thorburn, for the *International Medical Annual* for 1896. (Plates VI., VII., and VIII.)

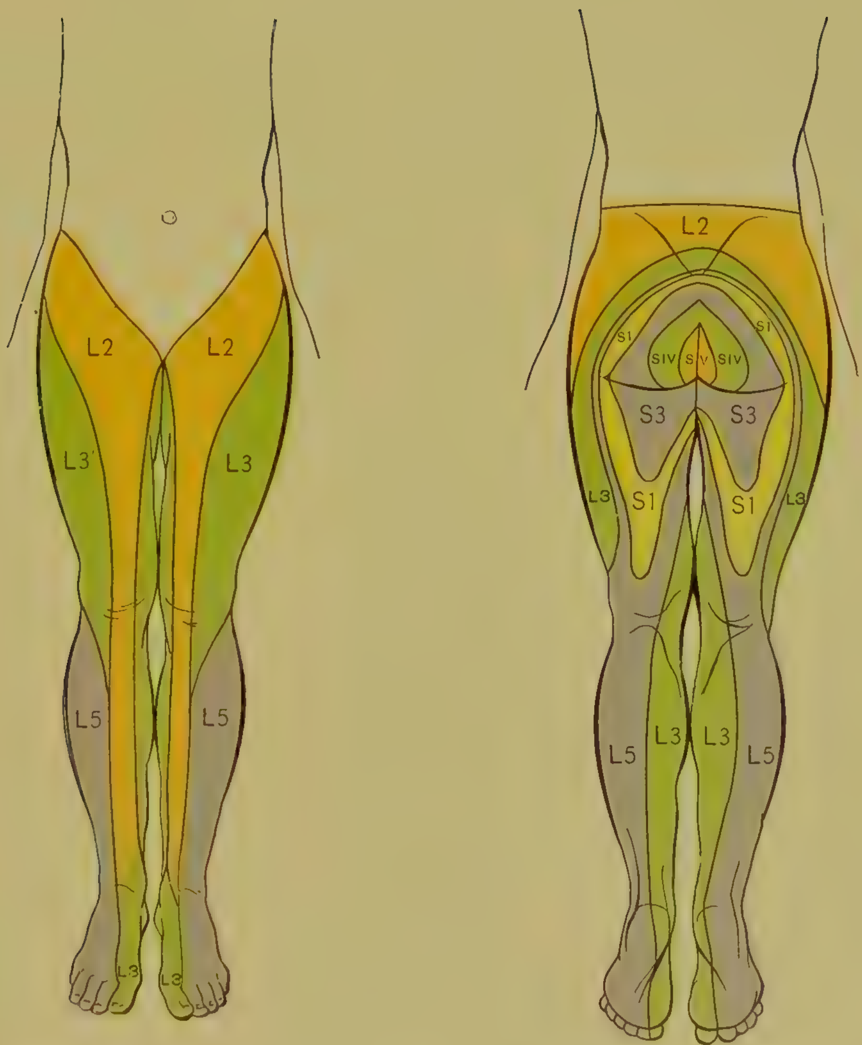
Anæsthesia of the skin in any part of the body may not only be due to cerebral or spinal lesions, but also to neuritis or inflammation of a nerve-trunk, or to some injury which prevents its functional activity by pressure, bruising, or cutting. As a rule, loss of sensation from neuritis occurs late in the disease, hyperæsthesia or paræsthesias being the earlier manifestations; but in some cases these are absent and anæsthesia begins at once. The

PLATE VII.



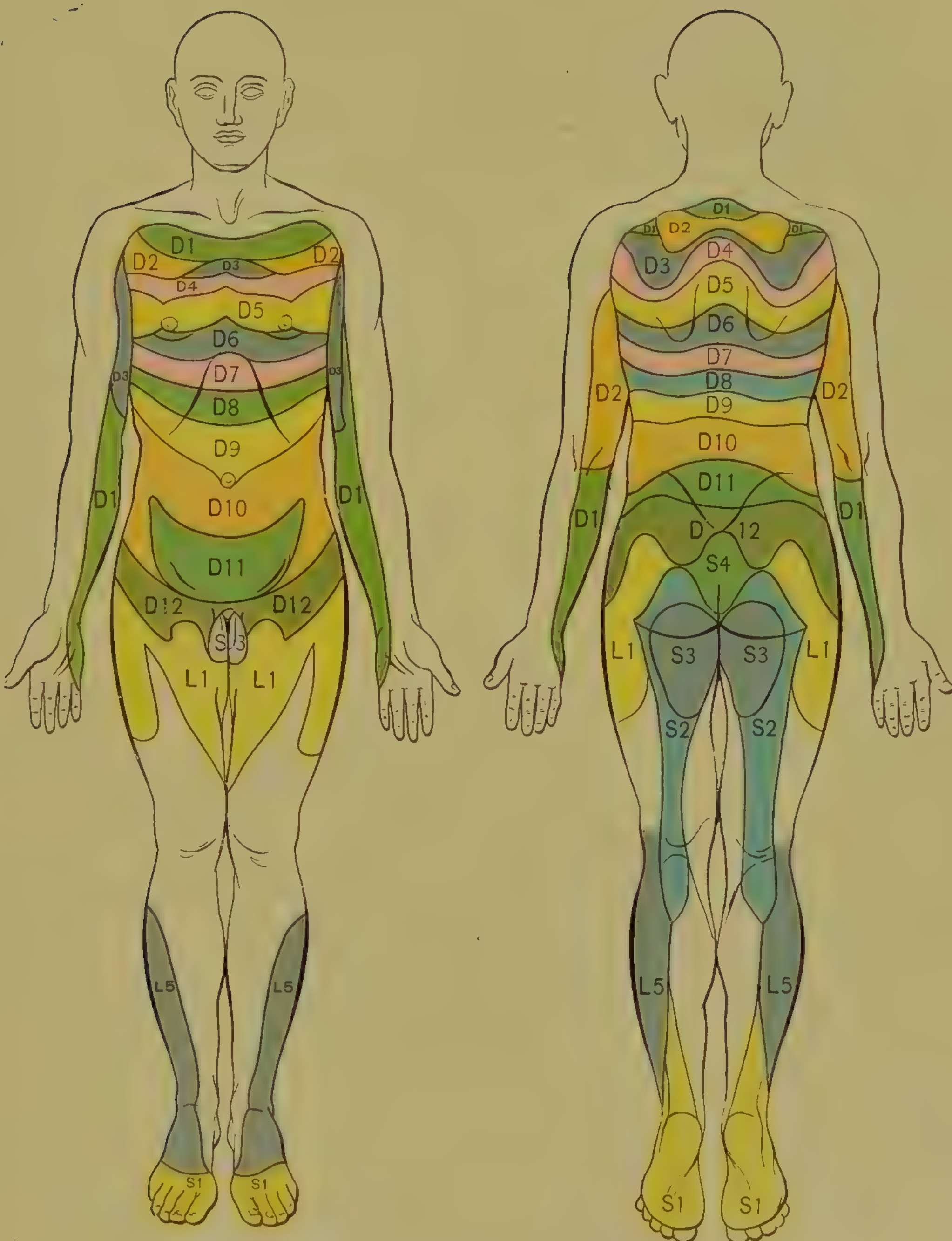
Cervical Roots are represented by the letter C, Dorsal Roots by the letter D, and Lumbar Roots by the letter L. (Chart after Thorburn.)

PLATE VIII.



Cervical Roots are represented by the Letter C, Dorsal Roots by the Letter D, and Lumbar Roots by the Letter L. (Chart after H[illegible].)

PLATE IX.

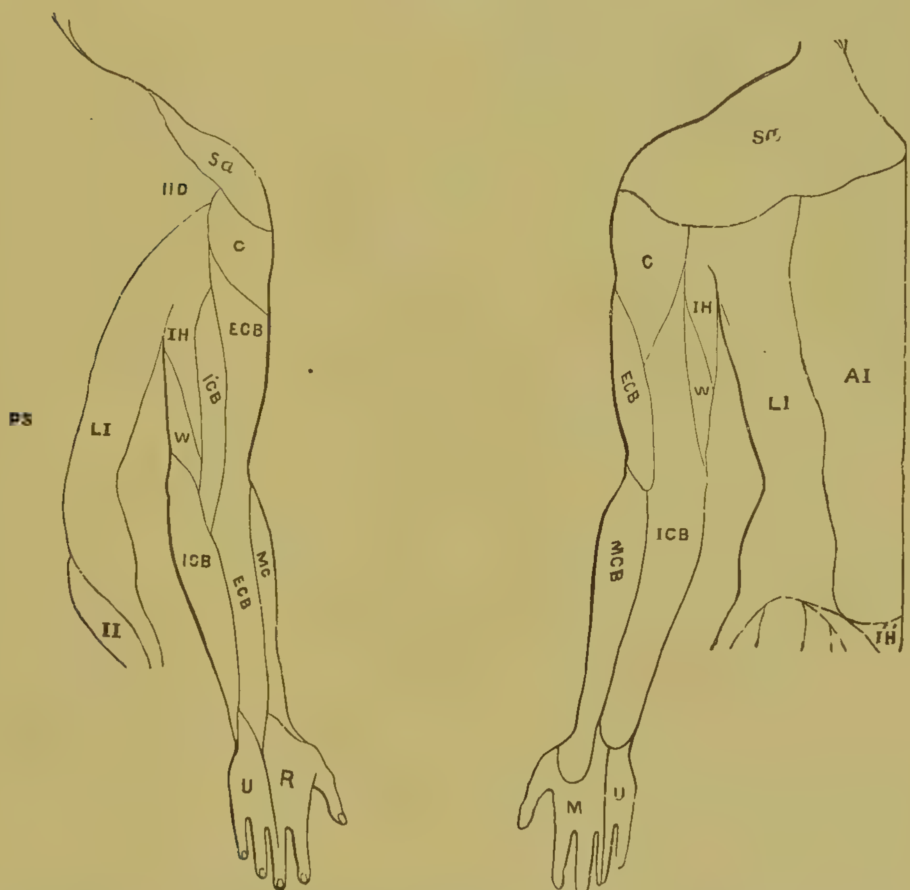


Cervical Roots are represented by the letter C, Dorsal Roots by the letter D, and Lumbar Roots by the letter L. (Chart after Starr.)

tion is not disturbed, even though the divided nerve be the sensory as well as the motor supply to the part.

It is well to remember also that sensory disturbances of the skin following injuries of nerves are often not nearly so great as the motor disturbance, even where there is no sensory transmission by anastomosis, and where they are present they usually disappear, more rapidly than the motor loss, as recovery takes place.

FIG. 101.

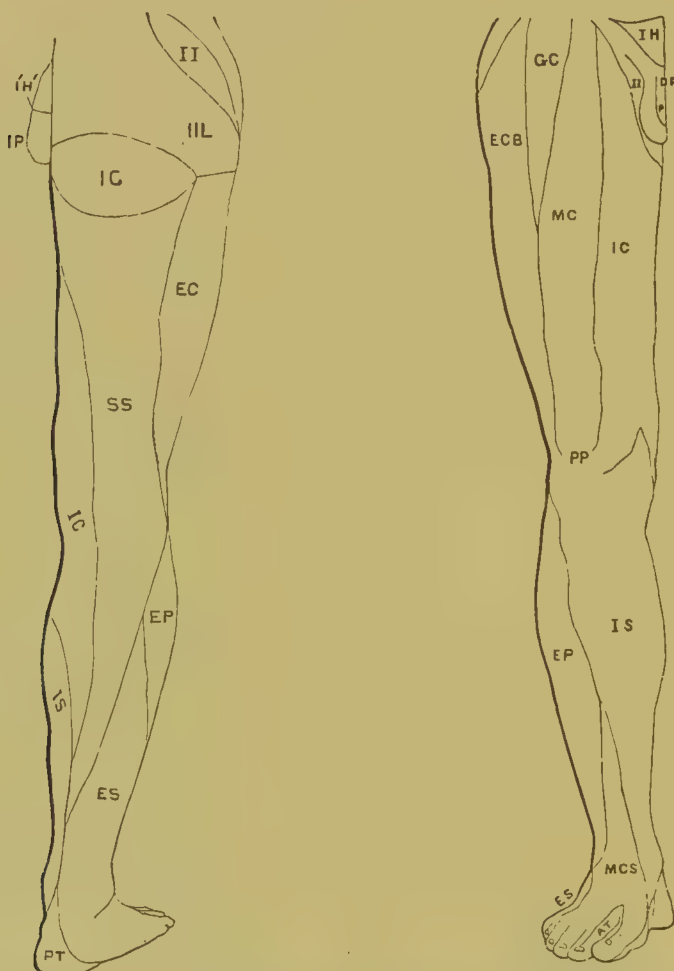


Cutaneous nerve of the trunk, and upper extremity. (FOWLER.) SA. Supraclavicular nerve. IID. Second dorsal. PS. Posterior branches of the spinal nerves. LI. Lateral branches of the intercostal nerves. AI. Anterior branches of the intercostal nerves. C. Circumflex nerve. IH. Intercostal humeral. W. Nerve of Wrisberg. I'CB. Internal cutaneous branch of musculo-spiral nerve. ECB. External cutaneous branch of musculo-spiral nerve. ICB. Internal cutaneous nerve. MC. Musculo-cutaneous nerve. R. Radial nerve. U. Ulnar nerve. M. Median nerve.

The following facts are, therefore, of diagnostic interest. If the anæsthesia is found to be due to a neuritis and to involve the palmar surface of the thumb, fore, and middle finger, the median nerve is probably the one at fault (Figs. 97 and 98), and the area may even include in rare instances the back of these fingers at their

bases and the half of the third finger nearest the thumb. When there is disturbance of sensation in the ulnar side of the ring-finger and in the skin of the little finger there may be ulnar neuritis (Figs. 99 and 100). (See also chapter on Hand.) The nerve-supply of the skin of the entire upper extremity is well seen in Fig. 101.

FIG. 102.



Cutaneous nerves of the lower extremity. (FOWLER.) Lumbar plexus. IH. Ilio-hypogastric nerve. II. Ilio-inguinal. IIL. Second lumbar nerve. GC. Genito-crural. EC. External cutaneous. MC. Middle cutaneous. IC. Internal cutaneous. IS. Internal saphenous. PP. Plexus patellæ. Sacral plexus. DP. Dorsalis penis of pudic. IP. Inferior hemorrhoidal of pudic. P. Superficial perineal of pudic and inferior prudendal of small sciatic. IG. Inferior gluteal of small sciatic. SS. Small sciatic. EP. Branches from external popliteal. ES. External saphenous. MCS. Musculo-cutaneous. AT. Branches of anterior tibial. PT. Branches of posterior tibial.

The development of sensory disturbances in the feet, resulting from neuritis, are as follows: When there is perverted sensation of the inner side of the foot from the tip of the big toe to the heel, and thence up the inside of the calf to the knee, the nerve

involved is the long or internal saphenous. When the dorsal surface of the foot has its cutaneous sense disturbed the nerve involved is the musculo-cutaneous, a branch of the external popliteal. Disturbance of sensation on the outer side of the foot and calf indicates failure of function in the external saphenous, which is composed of the cutaneous branches of the external and internal popliteal nerves. Disturbed sensation on the posterior surface of the calf also indicates trouble in the external saphenous nerve and communicans peronei, while when the sensation of the skin of the heel is disturbed the plantar cutaneous nerve, a branch of the posterior tibial, is involved.

In the skin of the thigh the anterior surface is supplied by the middle cutaneous nerve, which is a branch of the anterior crural; on the inner side by the internal cutaneous, also a branch of the anterior crural; and on the outer side by the external cutaneous, which arises from the second and third lumbar nerves. Laterally the external cutaneous gives the supply. Posteriorly the small sciatic gives the nerve-supply to the skin.

Anæsthesia of the greater portion of the skin of the thigh, except in a narrow strip on the back part and in the area supplied by the internal saphenous nerve, often occurs as the result of paralysis of the anterior crural nerve, arising from pelvic tumors, psoas abscess, and vertebral disease.

Facial anæsthesia and its diagnostic meaning are still to be considered. When it occurs it indicates that the fifth nerve, or its nucleus, is involved.

If the area be that of the forehead, upper eyelid, the conjunctiva and the nostril, the ophthalmic branch of the fifth nerve is at fault, and the lesion is probably at the sphenoidal fissure or within the orbit, and reflex winking of the eye no longer takes place because the conjunctiva is anæsthetic.

If the skin of the upper part of the face is anæsthetic, the superior maxillary branch is involved; and if the skin of the temporal region, that of the jaw and the under lip is anæsthetic, the inferior maxillary branch is diseased. When both of these branches are paralyzed there is probably a tumor of the superior maxillary bone; and if the entire area of the three branches is anæsthetic, the Gasserian ganglion may be the part affected, and this will be accompanied by trophic changes in the anæsthetic parts. The most common cause of anæsthesia of the trifacial is, however, neuritis.

Romberg makes the following differential statements :

a. The more the anæsthesia is confined to single filaments of the trigeminus, the more peripheral the seat of the cause will be found to be.

b. If the loss of sensation affects a portion of the facial surface, together with the corresponding faucial cavity, the disease may be assumed to involve the sensory fibres of the fifth pair before they separate to be distributed to their respective destinations ; in other words, a main division must be affected before or after its passage through the cranium.

c. When the entire sensory tract of the fifth nerve has lost its power, and there are at the same time derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerve in its immediate vicinty, is the seat of the disease.

d. If the anæsthesia of the fifth nerve is complicated with disturbed functions of adjoining cerebral nerves, it may be assumed that the cause is seated at the base of the brain.

Anæsthestic patches on the skin may be due to leprosy and syringomyelia, but in the former disease the macular patches are present or there may be found evidences of their previous existence in areas of skin, especially on the back, thighs, and calves, which are paler than normal and in which the sensation is partially blunted.

Rarely the anæsthesia of leprosy may be confused with that of Morvan's disease, and it may require a search for the lepra bacillus to separate them.

Other Disturbances of Sensation than Anæsthesia. The other disturbances of sensation of the skin than anæsthesia, which are usually subjective rather than objective, are paræsthesia, hyperæsthesia, and analgesia.

Paræsthesia or numbness, tingling, and burning, is seen in nearly all those cases in which anæsthesia ultimately develops as a result of organic lesions. When a patient complains that he cannot feel the contact of clothing about his feet and legs, or that the feet when he walks feel as if wrapped in some thick material, or as if he were walking on moss, or that the soles of his feet feel as if they were numb, and at the same time tickled by ants walking over them, the characteristic sensory disturbance of the skin seen in locomotor ataxia is present. Often there is tingling or numbness of the fingers, particularly the ring and little finger, and a sensation as if a girdle is about the patient is common. These are the subjective disturbances of sensation in tabes dorsalis, and, as they are often the earliest mani-

festations of the disease, are of great diagnostic importance. The objective sensory perversions consist in the discovery by the physician, when studying the sensibility of the skin, of areas of anæsthesia, analgesia, and hyperæsthesia, which are usually bilateral. Belmont has stated that we also find these areas in spinal syphilis, either on one or on both sides. Numbness, tingling, and formications affecting the skin are also often early symptoms of brain-tumor in the area supplying the affected part, and this possibility is increased if there is associated spasm. The actual objective sensibility of the skin may be preserved for some time after these symptoms appear, or it may be impaired almost at the outset, owing to the involvement of all or part of the sensory tracts in the cord. Similar symptoms are often seen in the early stages of myelitis. They are very frequently seen after injuries to nerves, and severe tingling in its acute variety occurs when the "funny bone" of the elbow is knocked against an object, owing to bruising the nerve. It is also seen in cases of aconite-poisoning and when the hands have been exposed to carbolic acid. Paræsthesias are also frequently seen in cases of neurasthenia.

Perversions of sensation in the skin sometimes take a curious form, as, for example, that known as allochiria, in which a sensory impulse in one hand is referred by the patient to the opposite hand. This is seen in tabes dorsalis, myelitis, multiple sclerosis, and hysteria. In other cases, as in paralysis agitans, this perversion takes place in the form of failure to distinguish heat and cold, and subjective sensations of extreme heat are felt. The part affected may actually have its temperature raised several degrees.

Very closely associated with the numbness of hysteria or neurasthenia, and lying between functional and organic disease of the nerves, is that condition called acroparæsthesia or waking numbness. This state is usually seen in women past middle life, but may occur in men. On waking in the morning marked formication and numbness of the fingers is present, which usually passes off as the day progresses, but as the condition becomes more marked they may last all day. While there is no anæsthesia, strictly speaking, the disturbed sense of touch renders sewing or performing any small act with the fingers almost impossible. These sensations may be confined to the area of one nerve, as the ulnar, or involve all the skin of the hands, or more rarely of the feet. General nervous excitability is generally associated with the local manifestations. Sometimes the scalp may be the area involved.

Acroparæsthesia is to be separated from the sensory disturbances of hysteria by its irregular outline, for generally in the latter disease the areas are distinctly outlined, by the fact that the hysterical condition is usually unilateral and by the absence of the characteristic general hysterical symptoms. From organic disease it is separated by the absence of the signs of neuritis about to be described, by the absence of tenderness, pain, and loss of power. From cerebral or spinal disease it is separated by the absence of symptoms produced by lesions in these parts, and by the fact that in both these instances there is paralysis of motion in association with the sensory disturbance, and in the case of spinal lesions the symptoms are usually in the legs, while acroparæsthesia generally manifests itself in the hands.

Closely associated with paræsthesia, if not an actual form of it, is the "girdle sensation;" that is, the patient feels as if a tight belt was strapped around a limb on the trunk. This is seen as a prominent symptom in locomotor ataxia, myelitis, and tumors of the cord or its envelopes. When the lesion is in the lower cervical or dorsal region the sensation is in the chest or abdomen; but this relationship between the growth and the sensation of constriction is not always constant. (See chapter on the Feet and Legs.)

Hyperæsthesia of the skin is an important symptom of both hysteria and neurasthenia, and its discovery in association with the peculiar symptoms which occur in the former morbid state confirms a diagnosis most positively. The most important and curious of these hyperæsthesias are the so-called hysterogenous zones, or, in other words, areas involving the skin and subcutaneous parts, which possess great sensitiveness and which, when pressed upon, cause in many cases convulsive seizures of the hysterical type. Not only is this true, but in addition it is a noteworthy fact that after the nervous disturbance produced by this means is set in motion, a second pressure on the hysterogenous zone may arrest the seizure. These zones commonly exist over the ovaries, in the groin, somewhere about the periphery of the mammary glands, or upon the spine in the lumbar or dorsal region. (See chapter on Pain.)

The hyperæsthesia due to neurasthenia is to a great extent spinal in character, but the skin of the rest of the back, particularly over the great muscles on each side of the spine, may also be involved. Often the neurasthenic patient will complain that in brushing or combing his hair pain or extreme sensitiveness is developed upon

the scalp, and there may be tender areas on the chest. These areas in neurasthenics can hardly be confused, even by the careless, with the hyperæsthetic zones of hysteria, and the personal history and characteristics of the individual aid still further in separating the two conditions.

Hyperæsthesia of the skin, aside from that seen in hysteria and neurasthenia, occurs in peripheral neuritis and locomotor ataxia, the skin of the back being particularly tender in this disease, and is frequently seen in a zone extending a little above the anæsthetic areas of transverse myelitis, this hyperæsthetic area being soon rendered anæsthetic by the progress of the disease. Hyperæsthesia in the skin of the limbs is also rarely seen in myelitis, and when there is motor paralysis of one side and sensory paralysis of the other it is commonly found on the side on which motion is lost. A condition of excessive dermal hyperæsthesia is also present in cerebro-spinal meningitis, in which disease it is often a very early symptom. It usually appears first in the legs, then in the hands and arms, and, finally, the skin of the face and head become involved.

Hyperæsthesia of the skin occurs, often associated with skin eruptions, in that very rare condition called chronic leptomeningitis. Motor symptoms are nearly always present if the cord become involved.

Hyperæsthesia of the skin is considered by some authors to be, when found in association with other characteristic symptoms, almost pathognomonic of brain-tumor. It may be found on the scalp, over a large part of the body, or in the part which is paralyzed. It is also found during the convalescence of typhoid fever, and in relapsing fever. It also appears in the paralyzed side of persons suffering from hemiplegia, in the area supplied by a nerve suffering from neuralgia, particularly that of a migraine type, in the scalp of persons suffering from gout, and in the same area in women about the time of the menopause.

General tenderness of the skin or deeper tissues is quite frequently seen in cases of rickets, the child crying whenever it is moved, as if sore and tender, and tender spots often appear over the ribs in cases of pleurisy.

Sometimes in a neurotic girl about the time of puberty, or in a woman, one breast becomes exceedingly painful and tender and the skin of the breast becomes so hyperæsthetic that the slightest touch causes pain. The whole breast is, moreover, tender, and the move-

ment of the arm may be impossible, owing to pain thereby caused in the gland. This hysterical breast can be separated from the painful breast due to a tumor by the general diffuse character of the swelling, the failure to outline any distinct mass, the neurotic character of the patient, and the age of the individual.

The hyperæsthesia of chronic alcoholism may be both dermal and deep, and is well marked along the course of the peripheral nerves, particularly where they emerge from deeper stuctures. It is also seen in the neuritis of lead and arsenical poisoning.

Increased sensibility of the skin may follow the use of opium or ergot, and is met with in the course of, or as a sequel of, influenza, and in some cases of profound anæmia.

In some cases hyperæsthesia is an early sign of the onset of non-tuberculated leprosy, and will generally be found in the course of the ulnar or sciatic nerves in such cases.

A very interesting fact from a physiological and diagnostic point of view is that disease of the internal organs or viscera often produces areas of hyperæsthesia or tenderness upon the skin, which may in future be used to aid in the localization of the lesions. This subject has been well studied by Head (*Brain*, 1893 and 1894), from whose researches much information may be derived, but the results of which will have to be confirmed in many cases before they can be used as diagnostic guides. (See article on Pain.)

Pain in the skin is very various in its manifestations, and nearly always is due to functional nervous troubles. Duhring has noted a boring sensation in some cases. It should direct the physician's attention to the possibility of hysteria or tabes dorsalis.

Pruritus or intense itching of the skin may be due to contact with some irritant, but its presence, if persistent, particularly if widespread or near the genitals, should always raise a suspicion of diabetes mellitus, or chronic lead-poisoning, or gout, or chronic contracted kidney. Very rarely opium may produce a pruritus, and jaundice is nearly always accompanied by some itching. Pruritus about the anus is often due to piles.

Finally, one important point is to be remembered, viz., we cannot attempt to make a general diagnosis merely from a study of the areas of anæsthesia or other perverted sensibility of the skin in any case. The results obtained from studies of the sensation of the skin are only to be used as additions to the motor and other symptoms which will be found discussed under the chapters on the Limbs.

CHAPTER VIII.

THE THORAX AND ITS VISCERA.

The inspection of the normal and abnormal chest—Their topography—Alterations in the shape of the thorax—The rhythm of the respirations—The results of using inspection, palpation, percussion, and auscultation in health and disease—The characteristic signs and symptoms of the various diseases of the thoracic organs.

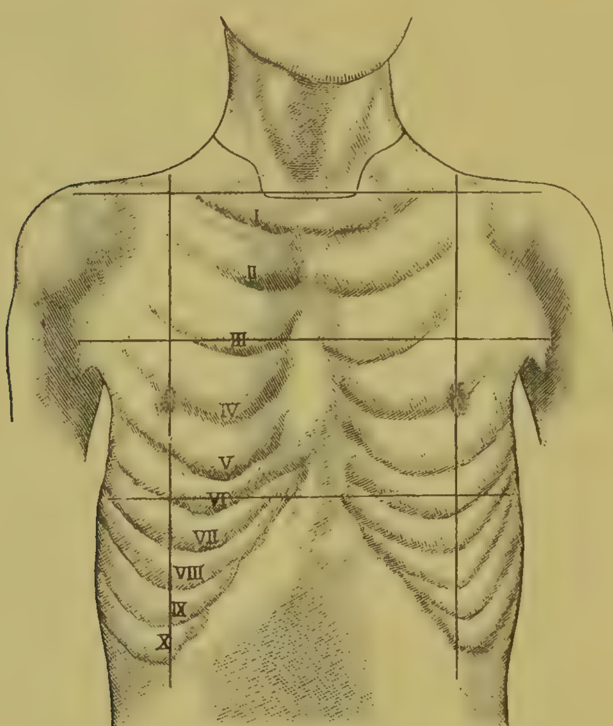
THE chief contents of the thoracic cavity consist of vital organs, which are unfortunately only too often subject to disease. A careful study of the signs associated with the normal functions of these parts is, therefore, of importance, as is also that of the symptoms indicating pathological changes. While it is true that in many instances patients present themselves to the physician with well-marked objective and subjective symptoms pointing to abnormalities in the organs of the chest, it is also a fact that in many others none of these signs exist, or at least in such an indefinite manner that the physician's attention is not attracted to them, and as a result important thoracic changes from the normal are overlooked or made light of. We base our diagnosis of the character of a case by the changes which we find in the surface of the thorax as to its contour and as to its movements, by the respiratory and cardiac sounds and the other physical signs about to be described.

Before we attempt to study the alterations produced by disease in this portion of the body we must, however, have a clear conception of the normal appearance of the chest and of the normal sounds which are produced within it.

INSPECTION OF THE NORMAL CHEST when free from clothing will reveal the fact that it is conical in form, the broader part of the cone being in the upper portion. Above the clavicles there is usually a slight depression (the supra-clavicular fossa), and below the clavicles, which may be somewhat prominent, there is also a slight convexity, which extends as far down as the fourth rib. This convexity varies considerably according to the muscular development of the individual, the formation of the bony portion of the chest-wall, and the deposit of fat in the subcutaneous tissues of the chest. The nipple is by no means as definite a landmark as is sometimes thought, as its position,

in respect to the ribs under it, varies greatly in different individuals; and it is still further altered in its position by the presence of much fat under it, or, again, in multiparous women owing to the relaxation of the breast. In the average adult male or virgin female the nipple is on a level with the fourth rib or fourth interspace. The ribs in a well-developed person are not prominent in the upper two-thirds of the chest, but in the lower third are more readily seen, particularly at the sides, because of their thin covering by muscles and the other subcutaneous tissues and the skin. The sternum in front and the spine behind are normally in the middle line. Over the top of the sternum is a depression called the episternal notch.

FIG. 103.



The regions of the anterior aspect of the chest. The Roman numerals indicate the ribs.

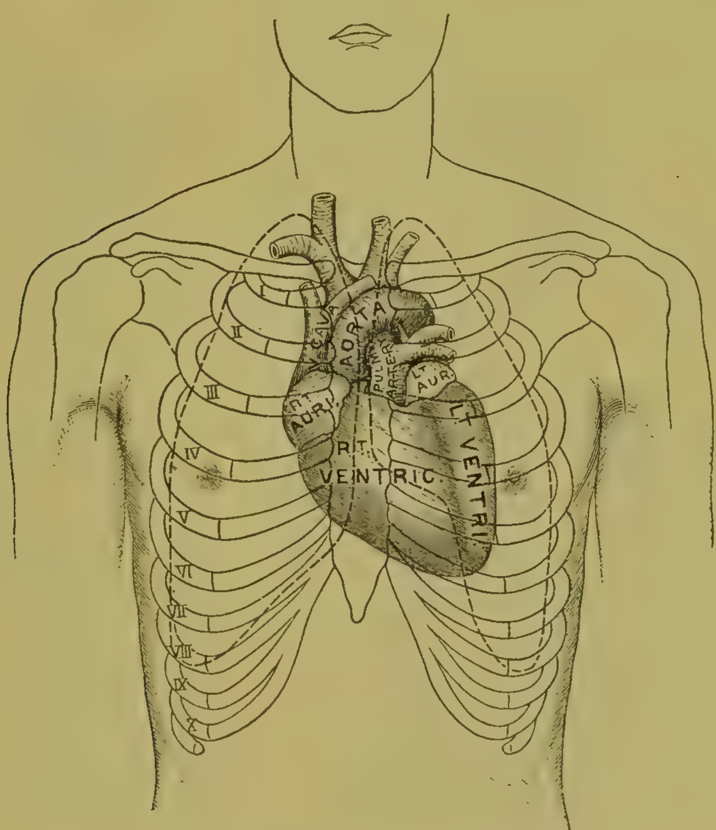
Lateral examination of the normal chest when compared to the front view will show that the antero-posterior diameter is less than the lateral diameter.

The surface of the chest anteriorly, posteriorly, and laterally, has been arbitrarily divided by imaginary lines into spaces, as shown in the accompanying figure. The lines running from the middle of the clavicles downward through the nipple are called the mammillary lines. (Fig. 103.) The parasternal line, not shown in the figure,

is a vertical line half-way between the middle of the sternum and the mammillary line; and a line running down the side from the axilla is called the mid-axillary line. These artificial divisions enable us to describe the locality of symptoms.

If we could see through the chest-wall, we would find that the lungs extend above the clavicles. Immediately back of the inner end of the clavicle is the beginning of the innominate vein, and back of this again the common carotid artery on the left side. On the right side the innominate artery bifurcates just behind the junction of the sternum and clavicle. The figure given below shows the relation of the cavities of the heart and its great vessels to the chest-wall. (Fig. 104.)

FIG. 104.



Position of heart in relation to ribs and sternum.

Anteriorly the lung extends as far as the sixth rib on the right, but the dome of the liver reaches to the level of the fourth interspace. On the left side the lung extends a little lower than on the right side. Laterally the lung on both sides extends to the ninth rib in the mid-axillary line. Posteriorly on the right side the lung

extends as low as the tenth rib and on the left side as low as the ninth.

Marked variations in the shape of the chest occur in healthy individuals without possessing any direct pathological significance. Thus, it is very common to see one shoulder slightly higher than the other, and, in the case of clerks or persons who work much at a desk, the left shoulder is very apt to be somewhat elevated. Occupations which cause the individual to assume certain positions, or to use certain muscles continually, also cause variations in the contour of the thorax.

INSPECTION OF THE ABNORMAL CHEST. The configurations of the chest which show a tendency to disease or the results of attacks of disease are numerous.

FIG. 105.

The alar chest of phthisis.

FIG. 106.

Side view of same patient.

The most familiar of these is the so-called phthisical chest, which has been called the "alar chest," because the scapulæ stand out from the back like wings. (Fig. 105.) The antero-posterior diameter, particularly in the upper two-thirds, is very slight, and instead of convexity in this part there may be flattening or hollowness. (Fig.

106.) This area scarcely moves on inspiration, but the lower thirds, which are bulging, move markedly with the respiratory efforts, as does also the epigastrium. The shoulders are very sloping, the neck, anteriorly, is receding at the episternal notch, but springs forward toward the Adam's apple and the chin. The ribs in the alar chest fall downward toward the belly from their points of origin, instead of coming forward in a normal curve. (Fig. 107.)

FIG. 107.

Phthisical chest.

If, on the other hand, the chest bulges anteriorly and posteriorly to such an extent that the antero-posterior diameter is greater than, or equal to, the lateral diameter, and if this bulging is fairly uniform, the shoulders being elevated, the back rounded, and the neck short in appearance from the raised shoulders, the patient is probably a sufferer from emphysema of the lungs. This chest is often called the "barrel-shaped chest." The chest-wall moves very little or not at all with the respiratory movements, which are chiefly diaphragmatic. (Fig. 108.)

Localized bulging of the chest results, in its most diffused type,

from the presence of chronic pleural effusion; bulging of a limited area also arises from cardiac hypertrophy, particularly that occurring in childhood; from aortic aneurism, causing bulging by pressure (Fig. 109); from pericardial effusion; and, finally, from mediastinal growths. Marked bulging over the lower part of the chest on the right side should cause us to look for some hepatic affection as well as to examine the lung, and, if the bulging is low down on the left side, to examine the spleen.

FIG. 108.

Boy fourteen years old with emphysema of lungs. Shows barrel-shaped chest.

FIG. 109.

Bulging of the chest-wall, with erosion of ribs from aortic and innominate aneurism.

Bulging or protrusion of the sternum, and the cartilaginous portions of the ribs attached to it, is called "pigeon breast," and is due either to rickets or to the presence of some obstruction to respiration of a more or less chronic character, during the time that the chest-wall has been soft and capable of being moulded. Sometimes on each side of the sternum, over the costal cartilages, there is seen a groove or depression as the result of rickets. In other cases a depression or groove extends from the ensiform cartilage back on either side toward the spine. This is called "Harrison's groove," and is

developed in children with poor bony systems, as the result of repeated attacks of asthma or other obstructive respiratory difficulty.

When examining the chests of children the physician will often notice a swelling of the tissues at the costo-cartilaginous junction, which look and feel to the touch like large beads under the skin. These beaded ribs are indicative of rickets, and are a manifestation of the general tendency to epiphyseal enlargement. This beading is usually most marked on the lower ribs.

Finally, unilateral bulging of the chest may be due to curvature of the spine, which part of the body should always be examined before a diagnosis as to deformity of the chest is attempted.

Shrinking of the chest in one part may be due to the contraction of old pleural adhesions, is sometimes seen over the diseased areas in pulmonary tuberculosis, and may be apparently present, but in reality due to wasting of the tissues covering the part.

While inspecting the surface of the chest the physician should also note the presence or absence of enlarged or pulsating bloodvessels on its surface or about the base of the neck. The cervical vessels are commonly seen to be distended in cases of advanced emphysema of the lungs and in chronic bronchitis, and systolic pulsation of the jugular veins indicates tricuspid regurgitation. Again, in cases of thoracic aneurism pressing upon the superior vena cava and innominate veins we find spongy venous masses above the clavicles, and the veins of the trunk and arms are apt to be engorged. Intra-thoracic growths produce similar symptoms.[1] Pulsation in the cervical vessels is also sometimes seen in cases of severe anæmia.

The shape and surface of the chest having been studied we can go further and learn much from its movements in respiration: first, from the rapidity of respiration; second, from the respiratory rhythm; third, from the character of the breathing; and, fourth, from the movements of the ribs.

When counting the respirations the physician should always endeavor to do so without letting the patient know that he is doing so, since it is difficult for many persons not to control their breathing when their attention is called to it. Generally the eye can detect the frequency of the breathing by simply watching the movement of the chest, or the information can be gained by resting the hand on the abdomen or thorax, while the wrist is also held and the doctor is

[1] See "Mediastinal Disease," by the author, Fothergillian Prize Essay of Medical Society of London for 1888.

apparently taking the pulse. In the newly born child in perfect health the respirations are often as high as forty-four, but in the adult male at rest they are usually about fourteen to sixteen per minute. During sleep the number may fall to eight or ten. The ratio of pulse to respiration is usually four to one, but in disease it may be one to one.

Rapid respirations not due to any recent sudden exertion are always indicative of respiratory trouble, primary or secondary. If the primary trouble be in the lung, it will probably be due to croupous pneumonia, catarrhal pneumonia, severe bronchitis, asthma, tuberculosis, pulmonary abscess, or tumors of the lungs. If it be due to secondary lesions in the lung, it may arise from pulmonary œdema due to nephritis, from congestion or hypostatic exudation as the result of a weak heart, from pulmonary embolism, from a pleural effusion which seriously interferes with the action of the lung or lungs, from growths in the mediastinum pressing upon bloodvessels and so causing exudation into the lungs or pleura, and from ascites or abdominal growths pressing upon the diaphragm. If the lungs be clear of trouble, then the difficulty may be present in the trachea or larynx, either as the result of spasmodic contraction of these passages or because they are occluded by growths, such as papilloma or malignant growth, inside or outside, which may act by pressure, thereby narrowing the tube. Any cause which interferes with the patient receiving the full amount of air usually inhaled causes rapid breathing in order that the loss of air may be compensated for by increased frequency of respiration.

There are, moreover, several other causes which affect the character of the respiration without affecting the larynx or lung-tissue directly or indirectly. These are fever, which acts as a respiratory stimulant, and excitement, nervous or mental, particularly that of hysterical patients. Again, apoplectic seizures, uræmia, and diabetic coma may be accompanied by rapid breathing.

The respirations are slowed or decreased in number by great obstruction to the entrance of air into the lungs from any cause; by the action of poisons made in the body, as the poison of uræmia or diabetes; by the effect of poisons swallowed or absorbed in other ways, notably opium, chloral, aconite, chloroform, or antimony.

The rhythm or relative time of inspiration, expiration, and the pause is in health in the mouth and trachea as follows: If 10 represents a complete respiratory cycle, inspiration is represented by 5,

expiration by 4, and the pause by 1. If there is difficulty in the entrance of air into the chest, as in spasmodic croup, the inspiration is much prolonged. This prolongation is also sometimes very marked in cases of paralysis of the posterior crico-arytenoid muscles. If there is difficulty in expelling the air, the expiration is prolonged, as in asthma or in emphysema.

Sometimes when the chest is flexible, as is that of a child, the inspiration is jerking when there is obstruction to breathing. This is due to the fact that the chest is forced into expansion by muscular effort, and at the same time is subjected to the external atmospheric pressure, while the air slowly enters the lung owing to the obstruction.

The most remarkable change in rhythm is the so-called Cheyne-Stokes breathing, in which the patient after a pause of several seconds begins to breathe with gradually increasing rapidity and depth, and then after reaching an acme of hurried respirations gradually decreases their rapidity and depth till they fade to nothing, when, after a pause, the same process is repeated. This breathing is seen commonly in apoplexy, in uræmia, in brain-tumor, in cerebro-spinal fever, in meningeal tuberculosis, in some rare cases of cardiac valvular disease, probably as the result of embolism, and in hæmaturic malarial fever. Rarely it occurs in cases of acute febrile disease, as typhoid fever, scarlet fever, pneumonia, whooping-cough, and puerperal septicæmia. It also may be met with in the course of diabetes. Its presence is an exceedingly bad prognostic sign, but cases of recovery after its onset have been observed, and Murri has reported a case in which Cheyne-Stokes breathing lasted forty days, and Sansom one in which it lasted one hundred and eight days. If the cause be an acute disease, recovery is more common after this symptom than if it be due to some chronic process with an acute exacerbation.

The function of breathing and the movements of the chest are closely associated. In men the respiratory movements chiefly affect the lower ribs and the abdominal walls, owing to the fact that as the diaphragm descends it pushes the abdominal contents downward, so causing abdominal bulging. In women, however, this is not so marked, and the breathing is chiefly costal, the upper part of the chest moving more than the lower (costal breathing). If abdominal breathing is absent in a man and is replaced by breathing of the costal type, we can be assured that the movements of his diaphragm are impaired by the pressure of fluid in the abdomen (ascites); by

peritonitis, causing fixation of the diaphragm, owing to pain; by the presence of large growths in the abdomen or by great enlargement of the liver and spleen. Other possible causes would be a subphrenic abscess or a greatly enlarged cystic kidney, or hydronephrosis.

If the costal breathing of a woman is absent, there is nearly always some pulmonary cause for it, such as faulty development, or, if due to disease, its absence arises most commonly from tuberculosis or pleurisy, or old pleural adhesions which bind down the chest-wall.

Labored breathing (dyspnœa) is seen in all cases in which the blood cannot be provided with sufficient oxygen owing to obstruction to the entrance of air into the chest, to spasm of the bronchioles, or to the occluding of the air-vesicles by any form of exudate, croupous, catarrhal, or serous. These conditions may be primary or secondary to disease elsewhere, as in uræmia or cardiac disease. Inspection of the chest in such a case shows great activity of the accessory respiratory muscles, such as the sterno-mastoids, the scaleni, the pectorals, and the abdominal recti. The nostrils are dilated and the face anxious. The posture of the patient is that of sitting up in bed.

Finally, we have to notice the extent of the chest-movements. These are very slight in the characteristic chest of a person having a tendency to tuberculosis, and in the barrel-shaped and rigid chest of emphysema of the lungs. Deficient expansion on inspiration is not only a predisposing cause for lung disease, but an important diagnostic sign. When one side of the chest moves more than the other to a considerable extent, we suspect, in the side which moves slightly, a pneumonia, a pleuritis, a pleuritic effusion or adhesion, or tubercular consolidation, provided that the patient has not naturally a greater development on one side than the other, or has not pursued a trade or occupation causing unilateral hypertrophy.

In this connection should be mentioned the "*wavy breathing*," seen most commonly in pneumonia, a condition in which inspiration and expiration do not seem to occur regularly or evenly all over the chest, one part filling or emptying a moment before the other. This usually indicates a grave pulmonary condition.

PALPATION of the chest reveals alterations in its contour and in its elasticity. It will also reveal the ability of the thoracic viscera and the chest-wall to transmit vibrations produced by the voice (vocal fremitus). This so-called vocal fremitus depends upon the fact that below the vocal bands lies a column of air which reaches to the vesicular portions of the lung, so that when an individual speaks this

column of air is set into vibration and these vibrations are in turn transmitted to the chest-wall. Of course, a chest-wall greatly thickened by fat or by highly developed muscles will not transmit these vibrations as readily as a thin chest-wall; but aside from these causes of variations in fremitus in health we have a number of causes in disease which greatly modify vocal fremitus. It must be remembered, too, that this vibration is more marked in men than in women and children, because the voice of a man is so much louder and has greater volume. Vocal fremitus is also greater on the right side than the left, because the principal bronchus supplying this lung is larger than that of the left side, is joined to the trachea at a less acute angle, and is nearer the vertebral column; and, again, as recently emphasized by Cary, the bronchus going to the right upper lobe is given off at a point very near the origin of the right bronchus, and in many cases "fully two and a half inches above the corresponding left bronchial tube." Sometimes this upper tube comes off the trachea directly.

The conditions of disease which cause a decrease in vocal fremitus are pleural effusions of any kind, which not only cut off the transmission of sound, but by their contact prevent vibration of the chest-wall; pneumothorax, which causes collapse of the transmitting medium, the lung; any condition which causes occlusion of a large bronchus, such as tumor or a large mass of mucus, and great pleural thickening. When the vocal fremitus is increased it is an indication of pneumonia, of tubercular thickening or consolidation of the lung, of the presence of cavities or of tumor in the thorax touching the chest-wall. Fremitus is increased in these conditions because the consolidated lung transmits the vibrations of the air in the bronchial tubes to the chest-wall, or, in the case of a cavity, the sound is transmitted directly to it, and it there causes so great a vibration of the air in the hollow space that the vibration of the chest-wall is marked. (In this connection, see part of this chapter on Auscultation.)

Palpation of the chest-wall will also give information as to the position and character of the cardiac pulsations. Thus, the apex-beat of the heart in persons standing erect will usually be felt, in persons not inordinately fat and who are healthy, between the fifth and sixth ribs, about two inches to the left of the sternum. (See Fig. 126.) If the apex-beat is below this level, its depression may be due to enlargement of the heart (hypertrophy or dilatation), to effusion in the pericardial sac or pleural cavity on the left side, to

pulmonary emphysema causing abnormal descent of the diaphragm, and with it cardiac hypertrophy. Sometimes tumors in the chest produce a similar depression. On the other hand, if the apex-beat of the heart is felt above the fifth interspace, the heart may be raised by pericardial adhesions following inflammation, by pleural adhesions or effusions, by abdominal effusion (ascites), by tumors, distention of the colon by gas, and by great enlargement of the spleen. Displacement of the apex-beat to the left is generally associated with downward displacement, and is generally due to hypertrophy of the left ventricle, to pleural adhesions, and particularly to pleural effusion on the right side. Displacements to the right are due to hypertrophy and dilatation of the right ventricle, so that the apex-beat is felt in the epigastrium or against the edge of the sternum. Pleural effusion on the left side may also cause this displacement. (See figures showing changes in cardiac area on pages 267 and 268.)

The area of the normal apex-beat is about one square inch. In disease this area often extends over several square inches, generally as the result of hypertrophy and dilatation of the ventricles.

The strength of the beat in health depends largely upon the depth of the chest and the thickness of its walls. In disease it is increased in hypertrophy of the heart and decreased in cases of feebleness of the heart-muscle, by effusions into the pericardium and the presence of pulmonary emphysema, which cause the projection of a part of the enlarged lung between the heart and the chest-wall.

Thrills felt in the chest-wall over the heart may be due to abnormalities in the blood-current when valvular disease or aneurism is present. We find thrills in the præcordium, or the neighborhood of the apex, in disease of the mitral valve, both regurgitant and obstructive; and thrills in the neighborhood of the second right costal cartilage indicate an aortic lesion, generally that of aortic stenosis, of aortitis, or of aortic aneurism. When thrills are felt in the tricuspid area, namely, in the midsternal region or a little to the right of it, the lesion is probably tricuspid regurgitation, as tricuspid obstruction is quite rare, or to aneurism of the descending part of the aorta.

In this connection we should remember the pulsation felt in the chest-wall in some cases of empyema. In nearly every instance this pulsation, when it occurs, is found on the left side. It is produced by the impulse of the heart against the effusion, and occurs in two forms: the internal, in which the effusion transmits a heaving impulse to the chest; and the external, in which there is a pulsating

tumor external to the chest-wall. Sometimes this is called "pulsating pleurisy."

Percussion of the chest is commonly performed by placing one finger, generally the middle one of the left hand, on the chest-wall and tapping it on the back with the tip of the bent finger of the right hand, the movement of the striking hand being entirely a wrist-movement. Sometimes percussion is made by directly striking the chest with the fingers or palm of the hand (direct percussion). Many physicians also employ a percussion-hammer with a rubber head and a pleximeter, or chest-piece, of ivory, celluloid, or glass. Glass is by far the best material for the chest-piece, as it does not produce a note of its own when struck by the hammer, as do the other materials. The disadvantage of this means of percussion is that the physician cannot determine the degree of resistance offered by the surface percussed, which is of the greatest service in many cases of doubtful character, as, for example, in a case in which pneumonia is suspected, and the results of the percussion will decide the diagnosis. Care should be taken in performing percussion: first, that similar points on the chest-wall on each side are carefully compared; second, that the finger which is applied to the chest is placed in the same relation to the ribs, or interspaces, on each side when it is struck; and, finally, in studying the effects of percussion the physician should always employ it both during forced inspiration and forced expiration, in order to determine the resonance of the chest with its full quota of air and when it has only residual air.

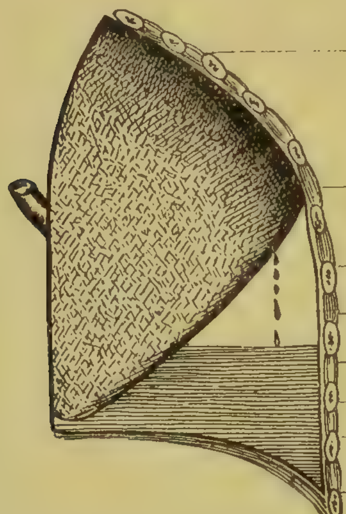
The resonance produced on percussion is due to three things: first, to the vibrations of the air in the lungs; second, to the vibrations of the chest-wall when it is struck; third, to the vibrations in the pleximeter placed on the chest. The last need only be considered as a factor when a piece of celluloid or ivory takes the place of the finger, for the finger itself does not vibrate enough to alter the note developed. The note produced by vibration of the chest-wall can also be excluded as of little importance unless the chest is very pliable and resilient, as in a thin child, and the blow be delivered very hard. The most important factor in the production of the percussion-note is that first named, viz., the vibration of the air in the chest caused by the blow delivered on the chest-wall. A large part of the percussion-note depends therefore upon the amount of air in the chest; the tension of the chest-wall; and the condition of the pulmonary tissues. The

sound produced when the healthy chest is percussed is called normal pulmonary resonance.

On percussing the right side of the chest anteriorly in the mammillary line we find in health normal pulmonary resonance as low as the fourth interspace or fifth rib, at which point the resonance begins to be impaired, so that at the sixth interspace or seventh rib we find the dulness. The area of partial and absolute hepatic dulness is shown in Fig. 140, in the chapter on the Abdomen.

Posteriorly we find on percussion that the normal pulmonary resonance begins as high as the suprascapular area, and ends as low as the tenth or eleventh rib. It is much less resonant as compared with the percussion-notes obtained from the anterior aspect of the

FIG. 110.



The condition of parts in hydro-pneumothorax from a perforation in the pleura. Metallic tinkling is represented by drops falling on the surface of the fluid. (GIBSON and RUSSELL.)

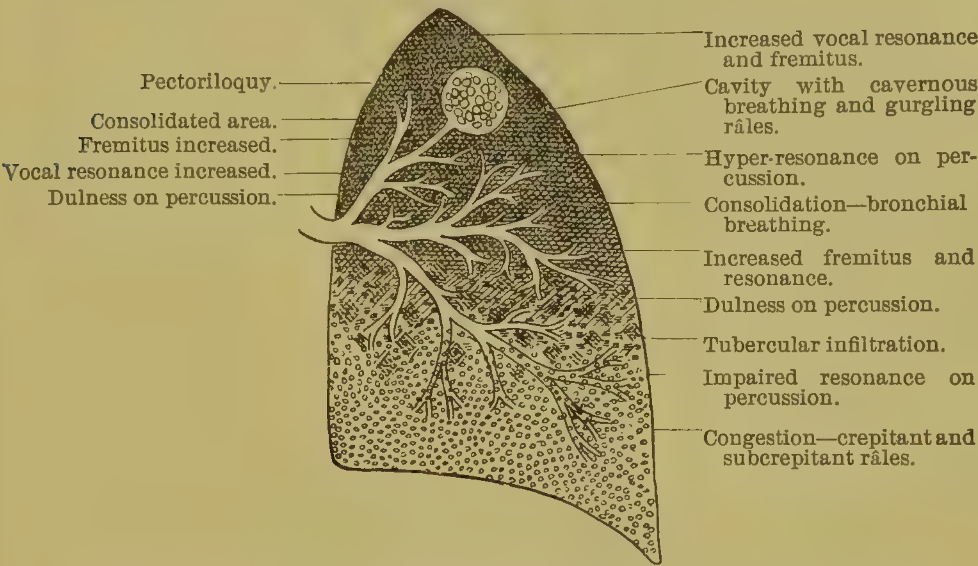
chest by reason of the thickness of the chest-wall and the presence of the scapulæ. For this reason pulmonary resonance is best developed posteriorly at the bases of the lungs below the scapulæ. Before percussing the back the patient should be made to lean forward and fold the arms in order to stretch the tissues and make them tense and as thin as possible.

We can divide the abnormal sounds produced by percussion into the tympanitic, the dull, and the flat. We can also develop by percussion of the chest in disease what is known as a "cracked-pot sound."

A tympanitic sound is best produced in its most typical form by percussing the epigastrium when the stomach and colon contain some

gas. We obtain this sound when the chest is percussed if there is present in the lung a large cavity, and also in pneumothorax (see

FIG. 111.



Phthisis at various stages in one lung, the physical signs depending on the stage. (GIBSON and RUSSELL.)

Fig. 110), in consolidation of the lung in some cases, and in some instances of adhesions or collapse of the lung-tissue.

If the cavity be in the lung itself, it must be of some size and be near the surface, and, if it communicates with a bronchus, the char-

FIG. 112.

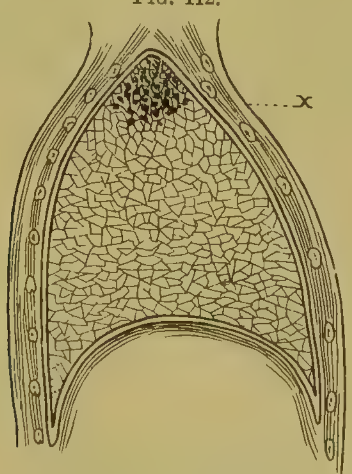


Diagram showing at x moderate dulness over tubercular infiltration. (GIBSON and RUSSELL.)

acter of the note will change when the mouth is closed or opened. (Fig. 111.) If the case be one of pneumothorax, with fluid in the chest, changes in the posture of the patient will greatly alter the char-

acter of the note. Consolidation of the lung, as in pneumonia and tuberculosis (Figs. 112 and 113), generally gives a dull rather than a tympanitic note, but if the consolidated area surrounds a very superficially placed bronchus, the percussion-stroke may produce vibration in the air in this tube, and this will cause a note, tympanitic in character, which varies as the mouth is closed or opened. Collapse of the lung causes a tympanitic note because the comparatively little air in the lung vibrates as a whole, its vibrations not being stopped as in

FIG. 113.

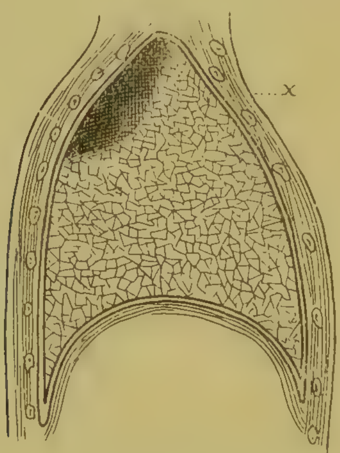


Diagram showing heightening of pitch anteriorly from consolidation posteriorly. The shaded part is the consolidated part; x indicates the position where the percussion-sound is raised in pitch. (GIBSON and RUSSELL.)

health by the tense septa and vesicular walls. It is best heard in cases of pleural effusion over the apex of the chest, into which the collapsed lung has been pushed by the effusion. This is sometimes called "skodaic resonance." If the compression is sufficient to consolidate the lung, the tympanitic note is lost. This note is not altered by opening and closing the mouth.

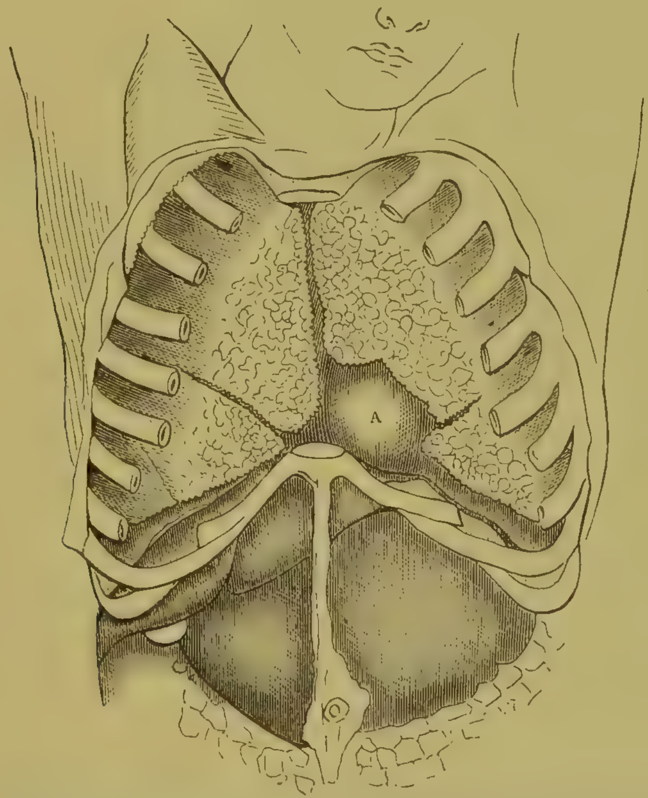
The "cracked-pot sound" is produced by the sudden expulsion of the air from a cavity through a small opening by the force of the percussion-stroke. It occurs on percussing a healthy child when its mouth is open, the air being forced by the blow from the lung through the glottis. In disease the cracked-pot sound most commonly results from the presence of a cavity in the lung. It may also be heard in cases of pneumothorax with a fistulous tract opening externally or into a bronchus; in a few cases of pleural effusion in thin-chested persons; and in rare instances before consolidation has occurred in pneumonia.

In cases of pleural effusion a flat note on percussion is heard over

the effusion, and it is of very much the same character as the sound elicited by percussion of the solid tissues of the thigh. (Fig. 110.)

Cardiac dulness. On percussing the chest anteriorly on the left side it will be found that the normal resonance is decreased by the presence of the heart. At the apex of the chest on this side percussion develops normal resonance, but as we descend in the line situated half-way between the mammary line and the midsternal line we find an impairment of resonance at the third rib, which becomes, in the next inch of descent, a very marked dulness, which is produced

FIG. 114.



Portion of heart uncovered by lungs. A shows the area of superficial cardiac dulness. (AITKEN.)

by the presence of a solid organ, the heart. The impairment of resonance is not complete at the upper border of the heart because of the fact that the edge of the lung intervenes between the heart and the chest-wall, and so the note which results on percussion is neither the normal resonance of the lung nor the dulness produced by the presence of the heart. (Fig. 114.) The outlines of the normal cardiac dulness on percussion are shown by the diagram which is appended, and they form what have been called the "cardiac triangles." (Fig. 115.)

The large triangle begins at the level of the second left costal cartilage and extends down the midsternal line to the level of the sixth costal cartilage. The base then extends to the apex-beat, normally situated in the fifth interspace just inside of the clavicular line. The hypothenuse of the triangle joins these points. In this area we have included the partial and total cardiac dulness.

FIG. 115.

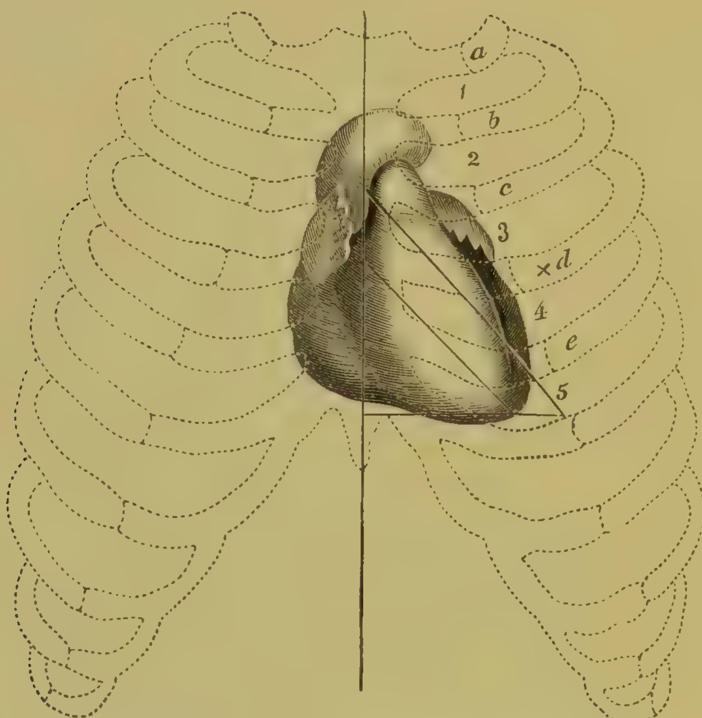
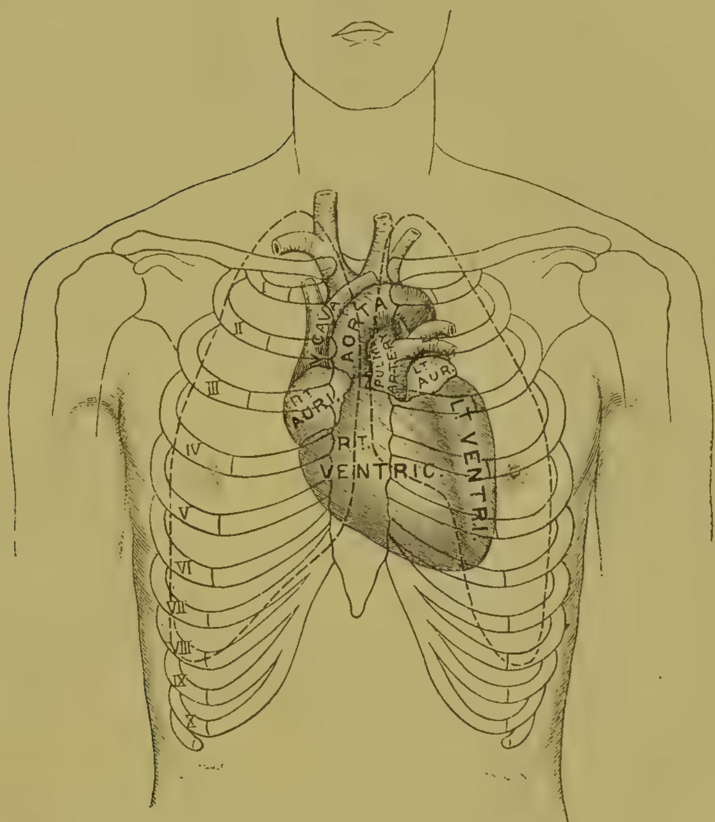


Diagram showing cardiac triangles. Compare this figure with Fig. 114.

The small cardiac triangle, which includes the absolute cardiac dulness, begins at the third costal cartilage and extends to the sixth. The base-line extends to within one and one-half inches of the nipple, and the hypothenuse joins this point with the third costal cartilage at the midsternal line. As will be seen from the diagram the borders of the heart really extend further than this, but are not near the chest-wall and are partly covered by lung-tissue. (Compare Fig. 114.)

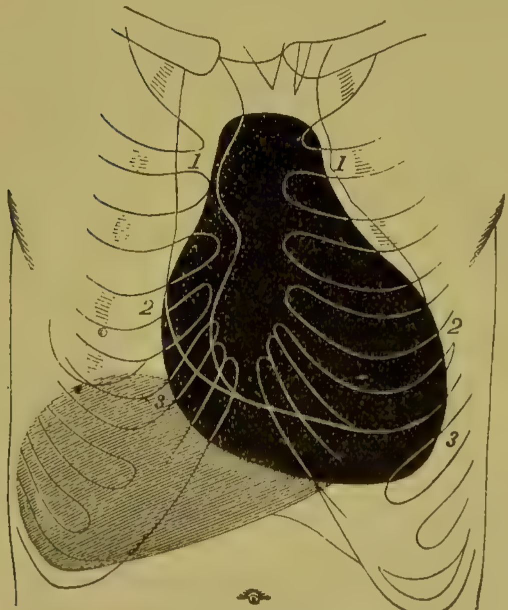
The greater part of the cardiac dulness on percussion is due in health to the presence of the right ventricle, which is nearest the chest-wall. The right auricle also is well forward, while the left ventricle only fringes the edge of dulness to the left. This is well shown in the accompanying diagram. (Fig. 116.)

FIG. 116.



Position of heart in relation to ribs and sternum.

FIG. 117.



Outline of percussion-dulness in a case of extensive pericardial effusion. (BYRAM-BRAMWELL, after SIBSON.)

When hypertrophy or dilatation of the heart occurs it will be found that the area of cardiac dulness extends to the right of the sternal line and to the left of the long side of the triangle, while the apex-beat is apt to be displaced downward and to the left. Great distortion of the triangles occurs as the result of pericardial effusion (Fig. 117), but in this case the heart-sounds will be distant on auscultation and the apex-beat very feeble or lost, whereas in hypertrophy they are exaggerated and the apex-beat forcible. The diagnosis of pericarditis, after the stage of dryness and friction-sound has passed by, is by no means as easily made as some of the text-books would make it appear. One of the most reliable signs of pericardial effusion is that of Rotch, namely, that any considerable dulness in the fifth right intercostal space means pericardial effusion, provided pulmonary consolidation and pleural effusions or adhesions are excluded. In dilatation of the heart the area of the apex-beat is usually diffuse, and the heart-sounds, while feeble, are clearly heard.

In this connection the following summary, prepared by Sansom, of the differential diagnosis between dulness due to pericarditis and that due to dilatation of heart may be of interest:

	Pericarditis with Effusion.	*Dilatation of the Heart.*
Outline of dulness .	Dulness pear-shaped, and enlargement chiefly upward.	Dulness not pear-shaped, and enlargement chiefly downward.
Rate of development of dulness	Often rapid, and then characteristic.	Usually very slow, though a rapid dilatation of the heart sometimes occurs.
Impulse and apex-beat	The impulse, when present, is in the third or fourth interspace; apex-beat tilted upward and outward, or effaced.	Impulse can usually be felt to the left of the lower end of the sternum or in the epigastrium.
Relation of dulness to left apex-beat .	Dulness may extend to the left of the apex-beat.	Dulness does not extend to the left of left apex-beat.
Pain over præcordia and tenderness in the epigastrium .	Often present.	Usually absent.
Pulsation in the veins of the neck . . .	May be present if endocarditis complicates.	Often present when right heart dilated.
Etiology	Usually acute, in course of acute rheumatism, cirrhotic Bright's disease, etc.	Usually chronic; often associated with chronic valvular lesions, fatty and fibroid degeneration.
Fever	Often present.	Absent unless from some complication.

The same author also tabulates the facts in the differential diagnosis between increased dulness due to pericarditis and hypertrophy of the heart as follows:

	Pericarditis with Effusion.	*Hypertrophy.*
Rate of development	Usually rapid.	Usually slow.
Impulse; apex-beat	Impulse, when present, is in the third or fourth left interspace, and is feeble; apex tilted upward and outward, or beat effaced.	Impulse powerful; if left ventricle hypertrophied, apex displaced downward and outward; if right ventricle hypertrophied, apex displaced downward and inward; beat may be in the epigastrium.
Pulse	Weak and quick; may be irregular.	Character of the pulse depends on the side of the heart which is hypertrophied and the cause of the hypertrophy. When left ventricle hypertrophied, and no aortic obstruction or mitral regurgitation, the pulse is large and powerful.

In emphysema of the lungs the cardiac triangles may be obliterated by the extension of the lung between the chest-wall and heart. They may also be distorted by reason of pleural effusions pressing the heart upward and to the right, or in the case of right-sided pleural effusion the heart may be pushed unduly to the left. Pneumothorax may cause similar results, or, again, old pleural adhesions and conditions may so displace the lungs or heart that the triangles cannot be found.

FIG. 118.

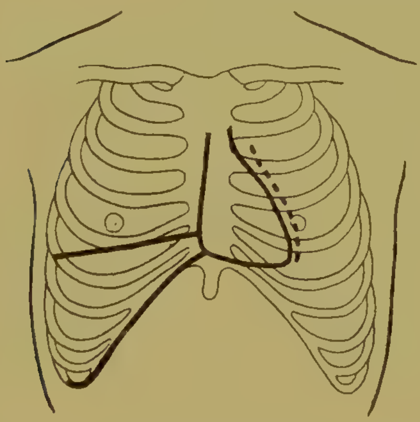


Diagram of the normal heart, the continuous line indicating the outline of the right, and the incomplete of the left cavities. (SANSOM.)

FIG. 119.

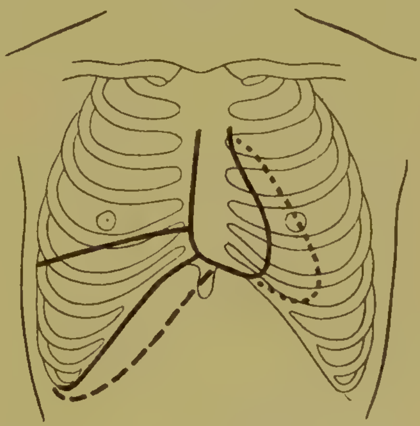


Diagram of the heart in aortic obstruction and regurgitation. The dotted lines indicate enlargement of the left cavities, especially the ventricle. The liver-area only slightly increased. (SANSOM.)

The various valvular and other lesions of the heart result in alteration in the size of the various cavities without the entire viscus being equally affected. Thus aortic regurgitation causes enormous enlargement of the left ventricle (dilatation and hypertrophy), and aortic stenosis also causes the same enlargement as a rule in less

degree. Mitral regurgitation causes hypertrophy and dilatation of the left ventricle and some enlargement of the left auricle, as does also mitral stenosis. Tricuspid regurgitation causes hypertrophy and dilatation of the right auricle and hypertrophy of the right ventricle, and mitral stenosis often has a similar influence over the right side of the heart by damming back the blood into the lungs and right side of the heart. The following figures from Sansom will illustrate the deformity of the cardiac triangles under these various conditions. (See Figs. 118, 119, 120, and 121.)

FIG. 120.

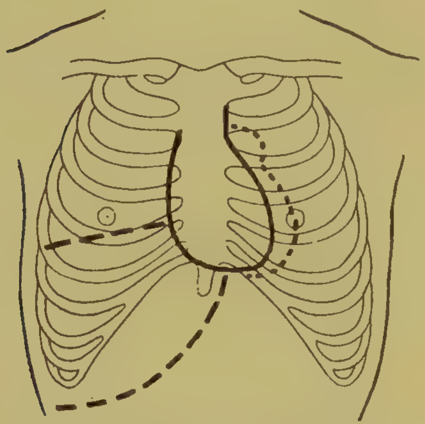


Diagram of the heart in regurgitation at the mitral orifice. The dotted lines indicate enlargement of the left auricle and the left ventricle, the continuous lines enlargement of the right ventricle and right auricle. The liver-area is much enlarged. (SANSOM.)

FIG. 121.

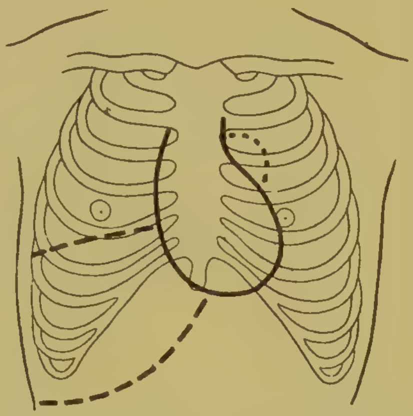


Diagram of the heart in obstruction at the mitral orifice. The dotted line indicates enlargement of the left auricle. The continuous lines show enlargement of the right cavities. The liver-area is much enlarged. (SANSOM.)

Finally, it is to be remembered that much information as to the thoracic organs may be gained by the sensation of resistance offered to the fingers on percussion. It is slight over cavities, greater over healthy lung-tissue, still greater over consolidations, and very great over effusions.

AUSCULTATION of the chest reveals in health two chief varieties of breath-sounds, namely, vesicular breathing and bronchial breathing. The first type is heard in its most typical form over the apices of the lungs anteriorly, the latter at the angles of the scapulæ posteriorly. We may listen to these sounds by placing the ear directly against the chest, or by the use of a single or a binaural stethoscope. The patient must be in an unconstrained position, as should be that of the physician, and if the ear is placed against the chest, or a single stethoscope is used, the face of the physician should always be turned

away from that of the patient, because the breath of a sick person is often very disagreeable and the breath of the doctor may be equally annoying to the patient. Care should be taken in the use of the stethoscope to see that the edge of the bell is in close contact with the chest-wall on its entire circumference.

The sounds which are heard in health in the chest on auscultation are respiratory and cardiac. The respiratory sounds consist, as already stated, in the vesicular murmur and the bronchial or blowing sounds, which are sometimes called tubular breathing. In the vesicles the air is subdivided into many minute parts, whereas in the bronchial tubes it moves along in a column. Whatever may be the actual cause of the production of normal vesicular breathing, we know that when it is present it signifies a healthy pulmonary parenchyma, and when absent one more or less diseased.

Bronchial breathing, normal in the bronchial tubes, becomes an abnormal sign when it is heard in an area in which vesicular breathing should be present, as will be shown shortly.

After determining the fact that the sounds of normal vesicular breathing are present in the anterior parts of the chest, or that those of bronchial breathing can be heard between the shoulders, we next take note as to the relative duration of the inspiratory and expiratory sounds. Normally in the perfectly healthy chest the ratio of the expiratory sound to the inspiratory sound is as one to three, although if the volume of air itself be measured the duration of expiration is six to five. In other words, so far as auscultation of the vesicular portion of the lung is concerned, inspiration is far longer than expiration. Just at this point we learn one of the most important points in the physical examination of the chest, namely, that while the expiratory sound may be entirely absent in health, any marked increase in its length and loudness, so that it equals or exceeds the inspiratory sound, is a sign indicative of some diseased state which impairs the elasticity of the lung, such as early tuberculosis, pneumonia, and emphysema.

The other variations in the vesicular respiratory sounds differing from those of health are harsh, or, as it is sometimes called, puerile breathing, and irregular breathing. In children, as the term "puerile breathing" indicates, the normal vesicular breathing is loud, clear, and harsh, because of the great elasticity of the lung and the thinness of the chest-wall. If it is exaggerated in a child or present in the area of normal vesicular breathing in adults, it usually indicates

some irritation of the bronchial mucous membrane. If it is found in the apices of the lungs in a marked degree and expiration is prolonged, it is an important and fairly sure sign of early pulmonary tuberculosis.

Sometimes physicians speak of "broncho-vesicular breathing," meaning a breath-sound consisting of both bronchial and vesicular sounds. It is sometimes heard in a healthy person when he breathes superficially, and in disease usually indicates early pneumonic changes or early tuberculosis of the lung. It is only of value as a diagnostic sign if localized in one part of the lung.

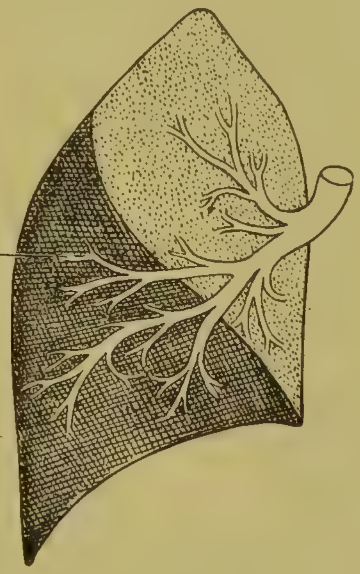
This harsh breathing of exudation and thickening differs from normal puerile breathing in this important particular, namely, that in the former expiration holds its normal ratio to inspiration, whereas in the latter it is greatly prolonged.

Irregular breathing occurs in the chest of a healthy sobbing child and in that of an hysterical woman, but it possesses pathological significance if it occurs when a full breath is taken, as it is often present as an early sign of incipient pulmonary phthisis.

Bronchial breathing in health is best heard in the posterior part of the chest, as already stated, between the scapulæ and the seventh cervical to the fourth dorsal vertebra. When this bronchial or tubular breathing is heard in other parts of the chest it is a sign of disease, for while the bronchial tubes are distributed to all parts of the lung, the breath-sound which is in them is masked by the sounds of vesicular breathing and muffled by the lung-tissue surrounding them. If this vesicular tissue becomes consolidated by disease, the vesicular murmur is lost and the solid lung transmits the bronchial sounds directly to the ear of the examiner. Bronchial or tubular breathing, or, as it is sometimes called, "blowing breathing," heard in the part of the lung in which vesicular breathing is normally heard, is therefore a sign of tubercular or pneumonic consolidation (Fig. 122) or of compression or collapse of the lung above a pleural effusion. Bronchial breathing is also heard in the area of the chest, in which vesicular sounds normally predominate, and in cases of cavity of the lung, because in such a lung the bronchial sound is transmitted directly to the cavity, and thence to the ear without being inflamed by the intervention of healthy lung-tissue. In other words, consolidated tissues and cavities transmit sound better than the normal vesicular portion of the lung, which is a combination of air and vesicular wall. If the cavity be large, we have a loud

sound developed by the transmission of the bronchial sound into its open space and by the passage of air through it. This is called cavernous breathing. If the cavity is not very large, or is peculiarly situated in relation to the supplying bronchus, we have what is called "amphoric breathing"—that is, a sound like that produced by blowing over the mouth of an empty bottle. This sound is also rarely heard in cases of pneumothorax in which the bronchial tubes, running near to the pleural cavity, transmit their sound to the air in the pleural space.

FIG. 122.



Pneumonia of the inferior lobe with the physical signs characteristic of consolidation. (GIBSON and RUSSELL.)

It is never to be forgotten that in examining the chest the two sides must be compared, since the well side often gives a standard for that affected by disease, and in doing so it must be remembered that disease not only modifies the signs in the lung in which the morbid process is situated, but also changes the normal signs. Thus pneumonia, or pleurisy or pleural effusion, causes a louder vesicular and bronchial breathing on the healthy side than is normal because this lung has to take in more air to make up for the loss of activity on the diseased side. Great care should therefore be exercised that the loud harsh breathing of the healthy part in such a condition is not mistaken for the harsh breathing of disease.

There are a number of other sounds heard in the chest in cases of disease of the air-passages. These consist in râles of various kinds, voice-sounds (vocal resonance), friction-sounds, and succussion notes or sounds.

Râles are divided into two chief classes, moist and dry. The moist are subdivided into the crepitant, or crackling, the fine bubbling, and the coarse bubbling. The dry are called sonorous, or sibilant and hissing. Sometimes the sonorous râles are called rhonchi. The fine crepitant râle is best imitated by pressing the thumb and finger-tip tightly together and then separating them while they are held near the ear. This râle is due to the separation of the vesicular walls, which have become adherent because of exudate. It occurs, of course, during the latter part of inspiration, and is an important sign of croupous pneumonia in its early stages before consolidation has occurred. It also is heard in cases of pulmonary collapse and œdema, but not always in any of these diseased conditions. Care should be taken that the fine râles, sometimes heard in the chest at the bases, posteriorly, in a person who has been long in one position in bed, are not thought to be indicative of pneumonia.

Fine bubbling râles occur chiefly in the smaller bronchioles and the coarse bubbling râles in the larger bronchioles, and they are caused by the passage of air through liquid or mucus. These are commonly heard in bronchitis and in pulmonary œdema in the lower parts of the chest, chiefly posteriorly. If such râles are heard anteriorly or in the area for vesicular breathing, they indicate the stage of resolution of a pneumonia, or if this disease has not been present, or is long gone by, they possess the serious import of breaking down of tissue from tuberculosis in the lung. Sometimes these râles are limited to inspiration or expiration. In convalescence from an attack of asthma they occur with a to-and-fro character, and are often musical or tinkling in character.

If a cavity has formed and liquid is in it, we may hear in the chest a peculiar hollow tinkling, called by Laennec "metallic tinkling." These sounds are sometimes heard over the stomach when this viscus is in motion and contains a little liquid and air.

There is another condition in which metallic tinkling is heard very clearly, and that is in hydro-pneumothorax. In this condition there is a continual dropping of liquid from the apex of the chest, or, more correctly, from the compressed lung in the apex of the chest, and as the drops fall through the air in the chest they strike the surface of the watery effusion with a tinkling sound. (Fig. 110.)

Râles are often removed or altered in character, if not crepitant, by coughing.

It has already been pointed out that dry râles may be divided into

the coarse or sonorous and the small or fine sibilant râles. They are produced by the passage of the air, in the large or smaller bronchial tubes, through partly inspissated and sticky mucus. If they are sonorous, the larger tubes are the part involved; if sibilant, the small bronchioles are affected.

It should not be forgotten that harsh breath-sounds made in the mouth or in the nose may cause the transmission of rough sounds or râles into the lungs, which will mislead the physician in his diagnosis if he thinks they arise in the pulmonary tissues.

Friction-sounds in the chest depend upon disease of the pleura or of the pericardium, generally the former. Normally the visceral and parietal layers of the serous membranes, which form the pericardium and pleuræ, present smooth surfaces which glide over one another noiselessly, but when they become roughened by disease a sound of friction is developed. Sometimes the friction-sound is so slight as to be almost inaudible, and again so harsh as to sound like a loud creaking, which can be not only heard, but will convey a sensation to the hand when it is placed on the chest. As a rule, friction-sounds due to pleuritis are best heard toward the close of inspiration and occur only in the early stage of the disease, ceasing with the development of the effusion and perhaps reappearing as the effusion is absorbed. The place where the sound is most audible is the axilla. If a friction-sound is heard at the apex of the chest, tuberculosis will often be the cause of its existence in this locality. Care should always be taken that fine râles are not mistaken for friction-sounds. They can be separated one from the other by the recollection of the facts that râles are modified by coughing, not affected by deep pressure on the chest-wall, and are usually well diffused, while the friction-sound is not modified by coughing, is intensified by pressure on the chest-wall, and is usually limited to a narrow area.

Pericardial friction-sound is, of course, heard best in the præcordium at the base of the heart—that is, at about the third rib. It is separated from pleural friction by the fact that it continues when the patient holds his breath.

Vocal resonance is closely allied to the sensation called vocal fremitus which is felt on palpation, as already described in this chapter. It is due to the transmission of the voice-sounds down the trachea into the bronchial tubes and bronchioles, and thence through the various portions of the lungs. If a stethoscope is placed in the episternal notch while the patient speaks, and the ear of the exam-

iner which is not closed by the instrument is closed by the pressure of his finger, the voice of the patient will be very clearly heard. If the stethoscope be placed between the vertebral column and the scapula posteriorly—in other words, over the bronchial tubes—the voice will also be clearly heard, but not as clearly as over the trachea, for two reasons: first, because the sound has already been divided into the different bronchial tubes, and, second, because the thickness of the chest-wall muffles it. If the stethoscope be placed over the anterior part of the chest toward the sides in the area of typical vesicular breathing, the sound of the voice will be still more modified, because the sound, like the air that conveys it, is now minutely subdivided, and the vibrations are decreased by the multitude of vesicular walls. Of course, the degree of transmission of vocal resonance is governed largely by the character of the voice, and for this reason it is more distinct in men than in women.

If the patient being examined is a man and has a well-developed voice, it is usually best to have him speak in a whisper, because the full volume of his voice is so great that it will be heard all over the chest and the nice differences between the transmission of the sound in the healthy lung and the diseased area cannot be distinguished. Usually we get the patient to speak by asking him to repeat his name or to count "one, two, three." The unemployed ear of the physician should always be closed, and the counting or speaking only be continued while the physician is actually listening to the chest.

In diseased states of the lung we find the resonance is increased by those changes which aid in the transmission of the sound and decreased by those changes which obstruct its transmission. As pointed out when speaking of vocal fremitus, a solidified lung and the opposite state, namely, a cavity, transmit sound better than healthy tissue, which is partly air and partly lung-tissue. We find, therefore, that the vocal resonance, or the sound of the voice of the patient when he speaks, is increased in pneumonia, in tubercular consolidation, and in cavity, and decreased in cases of emphysema, or in cases in which a pleural effusion separates the lung from the chest and deadens sound. (Fig. 107.) Vocal resonance, however, may be increased over pleural effusions, particularly the resonance of the whispered voice. This is called "Baccelli's sign," and Baccelli claims that it serves to separate serous effusions from purulent effu-

sions, because in his experience it is absent in the latter class of cases and present in the former.[1]

When a cavity is situated near the surface of the lung so that the sound of the voice is transmitted to it and from it through the chest-wall with unusual clearness, the sound so clearly heard is called "pectoriloquy." It is usually very marked over a cavity connected with a bronchial tube.

Sometimes, when the voice sounds through the chest-wall as if it were of a bleating character it is called "ægophony." It is usually heard at the angle of the scapula, near the margin of a pleural effusion and is supposed to be caused by compression and partial occlusion of a bronchus.

Finally, in pyo- or hydro-pneumothorax, if the ear be placed against the chest and the patient is shaken, we have developed a splashing or stopping sound, called "Hippocratic succussion." It is not always heard in these cases and may be developed when a large cavity in the lung is partly filled with liquid.

The healthy physical signs, and the variations from the normal signs met with in diseased conditions of the lungs, have now been discussed. The next step is to group these various signs with other characteristic symptoms in order that we may obtain a complete picture in the diagnosis of a given disease.

Let us suppose that a patient, previously in health or without any serious pulmonary complaint, is found, on a physical examination of his chest, to have rapid breathing, a somewhat anxious expression, a bright eye, and a dusky flush on one or both cheeks. Palpation discovers a hot, fevered skin, which is dry or more rarely moist, and increased vocal fremitus over both sides of the chest, more marked on one side than the other. Percussion reveals impairment of resonance over the area where fremitus was found most increased, and auscultation in this area shows bronchial breathing, fine crepitant râles, and increased vocal resonance. Under these circumstances we have before us the physical signs of *acute croupous pneumonia*. The pulse is apt to be rapid, but not as fast as the respiration would lead us to expect. The diagnosis is confirmed by the pain in the side affected, by the cough, the rusty, sticky sputum, and the history that the illness was sudden in onset and was initiated by a chill which may or may not have followed exposure.

[1] This sign is mentioned here for what it is worth. The writer has never been able to use it with success.

After a few days the râles disappear as consolidation becomes complete in the affected part, and the area which gave impaired resonance on percussion now gives a dull note, while the bronchial breathing in the affected part becomes more marked. The lips are apt to be attacked by herpes. With the fall of temperature, or crisis, which may be reached by the third to the ninth day, the râles return (*râles redux*) and become more and more loose, coarse, and moist, as resolution progresses, until the lung becomes entirely clear and only a slight roughening of the breath-sounds is to be heard. Bad symptoms in such a case are delirium, a feeble pulse, a feeble heart with distant heart-sounds, or one in which the action is labored and irregular. Prune-juice sputum, or, as the disease progresses, purulent sputum, are bad signs also. If the temperature falls to normal about the fifth day and then rises again, forming a pseudo-crisis, the attack will probably be prolonged.

The condition of croupous pneumonia cannot readily be confused with any other disease because of its characteristic symptoms, but catarrhal pneumonia and tuberculosis of the lung often are confused. In *catarrhal pneumonia* the patient usually presents a history of some illness. The disease rarely begins with the marked and startling symptoms of the croupous form, but is insidious and accompanied by a milder but more prolonged and constant fever. Percussion often will not give the positively dull note which can be elicited in croupous pneumonia, and only impairment of resonance may be developed. There is increased vocal fremitus on palpation and increased vocal resonance on auscultation; there are also increased bronchial breathing and more bronchial râles than in the croupous form, for the disease is a broncho-pneumonia, involving the bronchial tubes and vesicles. The signs are generally diffuse, very often heard best at the bases posteriorly, and clear tubular breathing, such as is heard in the croupous form, is rarely to be found. The sputum is not sticky or rusty; the fever does not end by crisis, but rather by lysis; and the lung returns to its normal state very slowly, its progress toward health often remaining almost stationary for weeks at a time.

The separation of these symptoms of catarrhal pneumonia from those of early pulmonary tuberculosis are practically impossible by the physical signs until the case has progressed to a well-advanced position. Often catarrhal pneumonia merges into the tubercular condition, and very often the diagnosis of catarrhal pneumonia proves

to have been made in a case in which the disease is really tuberculosis. We have to rest the diagnosis of tuberculosis chiefly on the family history, the personal history, the fact that recovery does not take place, and, more important than all, the presence of tubercle bacilli in the sputum, or yellow elastic fibres which indicate a breaking down of the lung-tissues.

If the malady be tubercular and progressive, we soon find in the chest and sputum signs which make the diagnosis clear. The chest on inspection does not move with costal breathing as much as is normal; the hand placed upon it feels, when the patient speaks, that there is not only increased fremitus but a bubbling feeling from coarse râles, and auscultation also reveals the signs of the breaking down of lung-tissue. Finally, when a cavity is developed the percussion-sound over it becomes high-pitched, and, if the cavity be large, almost tympanitic, although all around it dulness may be present. The breathing now becomes more tubular or amphoric, and vocal resonance may be increased to such an extent that bronchophony or pectoriloquy becomes marked even in that part of the lung in which in health the vesicular sounds are heard most typically. (Fig. 123.) Prolongation of expiration is also present, and sweats, irregular hectic fever, and great loss of flesh ensue.

FIG. 123.

Case of pulmonary cavity due to tuberculosis. The central ring is the area giving the physical signs of cavity, with cavernous breathing and whispering pectoriloquy, and the outer ring that of consolidation (dulness), with rapid breaking down of the lung-tissue (moist râles).

The history of the case and its symptoms are our chief means of separating pulmonary abscess from pulmonary tuberculosis with the development of cavity, for the physical signs are about the same. In cases of abscess we find that the patient has suffered from pneumonia or from pyæmia, with embolic infarction. In other cases discharges from the nose and throat entering the lungs produce such lesions. The symptoms of abscess, which separate it from cavity

due to tuberculosis, are as follows: In abscess the lesion exists in the lower lobe as a rule, while the tubercular cavity is usually found at the apex or in the upper lobe. The constitutional disturbance in abscess is often very slight, whereas in tuberculosis it is usually severe. In abscess the sputum is copious and purulent, and often coughed up in gushes, whereas in tuberculosis it is often scanty, and not markedly purulent as a rule. Again, in the last-named disease tubercle bacilli may be found, but they are absent in abscess.

If the patient has the signs of cavity of the lung, and in addition an exceedingly fetid breath, with great wasting, the case is probably one of pulmonary gangrene. Gangrene is usually found at the base of one lung, as is abscess. The sputum is usually brownish.

Bronchiectasis with fetid breath is occasionally met with, but the fetor after coughing is never so horribly strong as it is in cases of gangrene.

There are two areas in the lung often affected very early in pneumonia, particularly of the croupous type, and in pulmonary tuberculosis, which are apt to be overlooked, namely, the axilla, and the septum between the upper and middle lobe on the right side, an area only exposed to percussion and auscultation when the right hand of the patient is placed on top of his head in such a way that the angle of the scapula is drawn away from the vertebral line. (Fig. 124.) If this is done, the inner border of the scapula will approximate the line of the septum, and along this line there will often be found in tuberculosis of this portion of the lung marked dulness on percussion or, on auscultation, râles, and the other physical signs of consolidation even though the physician is unable to find elsewhere any evidence of local disease to account for the general systemic symptoms. Very often careful auscultation of the axillary area will also reveal signs not to be found elsewhere which account for the illness, such as those of pneumonia or pleurisy, for here, as a rule, the friction-sounds of the latter affection are best heard.

The physical signs of pulmonary œdema may develop suddenly as a result of an injury to the vagus, or in acute disease of the lungs. Generally, however, their onset is slow and insidious, but the rapid breathing, crepitant râles, the limitations of these signs to the lower part of the chest, combined with dulness on percussion, the absence of fever, the frothy sputum, and, it may be, the history of renal disease all point to the true state of affairs.

There is another state that gives dulness on percussion, crepitant

râles, and the other physical signs of pneumonia, namely, pulmonary congestion dependent upon the action of a feeble heart in the course of prolonged exhausting fevers; but the history of the illness, the feeble heart, and the development of these signs in the dependent parts of the chest effectually preclude the idea of any acute inflammatory process in the lung.

FIG. 124.

Area of dulness found in many cases of obscure pulmonary tuberculosis, when the arm is raised so that the scapula no longer covers the septum.

Finally, we frequently have after a pulmonary apoplexy an area of consolidation in the lung; but if this be the case, we also have, as a rule, a history of hæmoptysis. This condition is, however, comparatively rare.

To cite another form of thoracic disease, let us suppose that a healthy man is seized with pain in the thorax and a chill followed by fever. An examination of his thorax will reveal on inspection

deficient breathing on the affected side, which is fixed because of pain produced by the inflamed pleural surfaces moving over one another on inspiration. Exaggerated breathing will be found on the opposite side to compensate for this fixation, and auscultation on the painful side will reveal a friction-sound, probably best heard in the axilla. After these signs have existed some hours the second stage develops, and as effusion takes place we find that the friction-sound disappears and that the affected side, previously almost normally resonant, is beginning to become dull, and, finally, is flat on percussion at the most dependent part of the pleural sac, namely, at the base of the lungs posteriorly. This area of flatness on percussion gradually rises higher and higher until the effusion is completed. It extends anteriorly, and may be demonstrated as well here as it can be posteriorly and laterally, although, if the patient lies on his back or is partly recumbent, the entire anterior surface of the chest may be resonant, owing to the fluid leaving the front of the chest and going to the more dependent parts. In other words, in cases of non-sacculated serous pleural effusion changes in the position of the patient cause alterations in the area of flatness on percussion, unless the effusion is large enough to fill the chest entirely, when, of course, it is immovable. Inspection will show an increase in the size of the chest on the diseased side, with bulging of the intercostal spaces.

A curious yet important point in this connection is the fact that the line where flatness on percussion ceases at the top of the effusion posteriorly is wavy or sigmoid (S-shaped). Above the level of the effusion percussion over the compressed lung gives a somewhat hollow note or hyper-resonance, called "skodaic resonance," and the sense of resistance to the percussed finger is less at this point than over the effusion, where the resistance is great. In ausculting the chest in the area in which flatness has been developed by percussion very distant breath-sounds are audible, except in the back near the vertebral column, where there may be marked blowing breathing. If the patient speaks, there will be found loss of vocal resonance and of fremitus over the effusion, but, along the margin of the spine on the diseased side, there may be heard in some cases bronchophony, or even the bleating voice-sound called ægophony. Inspection and palpation will show the apex-beat of the heart displaced to the right and downward in cases of effusion into the left pleura, and to the left in cases of right-sided effusion. Again, if the effusion

be on the left side, it will be found on percussing "Traube's semilunar space," a space directly in the nipple-line and a little below the nipple, that the usual tympanitic resonance normally found in this area is extinguished through the downward pressure of the fluid.

If the effusion be accompanied by pneumothorax, we will find three sets of physical signs, namely, those of effusion, which will be at the lowest part of the chest; next above this an area in which percussion gives a clear tympanitic note due to the air in the pleural cavity; and above this the physical signs of the compressed lung in the apex of the chest-cavity. In this condition we may also hear succussion or splashing sounds, if the patient is shaken while the physician's ear is against the chest-wall, and the metallic tinkling, or dropping sounds, as the fluid falls from the top of the chest-cavity into the effusion. Again, we may use what has been called "dollar percussion." This consists in having an assistant place a silver dollar against the chest-wall on the diseased side anteriorly, and then the physician listens at the posterior aspect of the chest, with his unused ear closed by his finger. The assistant now strikes the silver dollar with the edge of another silver dollar. If the dollars be struck together below the surface of the effusion, very little of the metallic sound will be transmitted through the chest. If the dollars are struck together at the level of the layer of air, the sounds come through the chest-cavity with startling clearness; but if at the level of the lung, they are less clearly heard than at the level of the air, but more so than at the level of the effusion. The reasons for this are obvious, for the liquid prevents the transmission of the metallic sounds, as does also to some extent the compressed lung at the apex of the chest, whereas the space filled with air conveys the sounds directly to the ear.

Finally, if the effusion is absorbed by unaided nature, the area of flatness on percussion becomes less and less great from above downward, the expansion of the chest on inspiration increases, the interspaces cease to bulge, and the friction-sounds may return for a brief period.

If the effusion does not disappear, the physical signs of its existence persist; and if it becomes purulent, the patient is apt to lose flesh and strength, to have chills, fevers, and sweats, and to present all the evidences of an accumulation of pus in some part of the body. Particularly is this result apt to follow a pleurisy complicating one of the acute infectious diseases, such as scarlet fever, typhoid fever,

some instances of pneumonia, and in many cases in which tuberculosis is responsible for the illness.

Particular attention should be called to the possibility of pleural effusions coming on insidiously. There is probably no other massive pathological change anywhere in the body so often unsuspected or overlooked, and it is noteworthy that when pleural effusion is insidious in its onset and devoid of prodromes, that it is often due to an undiscovered tuberculosis, whether the exudate be found to be serous or purulent. Again, the fact that tubercle bacilli cannot be found in the effusion when it is aspirated in no way proves that the effusion is not tubercular in origin, since they are rarely found in the fluid even when tubercular pleurisy is most active.

If on aspirating the fluid in the chest it is found to be hemorrhagic in character, the cause may be one of the diseases which produce marked asthenia, notably carcinoma, nephritis, one of the acute infectious diseases in a malignant form, and tuberculosis. The cancer may or may not be in the chest. Rarely such an effusion occurs in otherwise healthy men without these causes. The possibility of the hemorrhagic effusion being due to a leaking aneurism, or to leakage from an ulcerated bloodvessel in tubercular disease of the lung, is to be remembered.

If after exposure to cold there is a sense of soreness in the chest, with more or less oppression and a hard cough, which seems to tear the bronchial tubes, the cough being without associated expectoration and the febrile movement but moderate, we suspect the presence of an acute bronchitis, a diagnosis which will be confirmed if we find the following physical signs :

There is marked roughening of the breath-sounds all over the chest, particularly over the bronchial tubes at the back, between the scapulæ, without any increase in vocal resonance and fremitus, or any impairment of resonance on percussion. As the disease progresses these rough sounds of harsh breathing give way to râles, which are at first fine and moist, then coarse and sonorous, as the second stage, or stage of secretion, develops ; and, finally, these decrease little by little, as health is approached and the mucus is expelled by coughing. Care should always be taken to determine in examining a case of suspected bronchitis that the symptoms are not due to a broncho-pneumonia.

Should the case become chronic the sounds of coarse, and more or less sonorous, râles will persist and will become constant. Such

cases usually become worse in winter and the sputum is sometimes very profuse (bronchorrhœa). The physician should always be careful in these cases to see to it that renal disease or a feeble heart is not the cause of the bronchial disorder. The health suffers but little in simple chronic bronchitis, but if bronchiectasis develops, it may be much impaired.

Under the name "putrid bronchitis" we have a state in which the sputum is foul and expelled in a liquid form, in which float little yellow plugs (Dittrich's plugs). This condition may end in pulmonary gangrene or cause metastatic abscess.

The presence of a barrel-shaped chest, with almost immovable walls and marked abdominal breathing, points to the presence of emphysema of the lungs, and this opinion is confirmed if on auscultation of the chest we find *marked prolongation of expiration,* diminished vocal resonance and fremitus, and increased resonance on percussion. The face is often quite cyanotic, the superficial veins of the neck turgescent, the abdominal respiratory movements abnormally great, and the superficial veins in the epigastrium enlarged. If bronchitis or bronchiectasis is associated with the emphysema, as is frequently the case, we find more or less marked râles all over the chest, particularly posteriorly. Sometimes a systolic murmur can be heard over the tricuspid area, due to regurgitation on the right side of the heart. Cardiac dulness is generally obliterated by the enlarged lung, and the apex-beat cannot be felt except in the neighborhood of the ensiform cartilage or in the epigastrium. Both the hepatic and splenic dulness are found to begin and extend lower than normal, owing to the expansion of the lung. We may also find accentuation of the second sound in the pulmonary artery. The tricuspid regurgitation usually develops from a damming up of the blood in the right ventricle.

When a patient is seized with a violent attack of dyspnœa its cause may be asthma, a foreign body in the air-passages, or laryngeal spasm.

If it is asthma, there will be labored breathing in which all the accessory muscles of respiration in the neck and trunk aid the ordinary respiratory muscles. The posture of the patient will usually be that of sitting up in bed and somewhat leaning forward. The face will be flushed, the vessels of the face and neck turgid, and the lips may be cyanotic. Often the patient, while sitting up, supports himself by resting on his hands, which are placed at his side in

order to raise his shoulders and fix the chest-walls for the contraction of the muscles which are endeavoring to drive out the air, for it is to be remembered that the respiratory difficulty in asthma depends more upon the fact that the patient cannot empty the lungs than upon the fact that he cannot fill them. As a matter of fact, they are too full of air which has been used.

Inspection not only shows these signs in asthma, but also reveals, in cases in which emphysema has not developed to such an extent as to cover the heart with the lung, that the apex-beat is diffused and the heart laboring. Palpation reveals little, except when coarse râles are present in large numbers, when some bubbling may be felt. Percussion usually gives an increased resonance because the chest is inordinately full of air, and auscultation reveals very loud blowing breathing, musical notes, or squeaking or creaking noises, both on inspiration and expiration. Finally, as secretion begins to be established musical and cooing râles may be heard in well-marked cases all over the chest before the ear is placed against the patient. At first these râles are heard chiefly on expiration, but very shortly they occur equally loudly on both inspiration and expiration. Toward the end of the attack coughing brings up a limited amount of sputum, which contains Curschmann's spirals and Charcot-Leyden crystals. (See chapter on Cough and Expectoration.)

As asthma is a symptom, not a disease in itself, the physician should always examine the nose, with the object of discovering some source of reflex irritation in the nasal mucous membrane, or the urine to discover whether renal disease is present, or the heart to discover if a cardiac lesion accounts for the symptoms. Sometimes gastric disorder is responsible for the attack.

Care should be taken that a catarrhal pneumonia developing after an attack of asthma is not overlooked until the patient is dangerously ill.

If on auscultating the chest we find it filled with musical and cooing râles, heard in every part, though most marked in the bronchial tubes, we can be fairly sure that an attack of asthma is about passing away; but if, on the other hand, the attack is beginning, the prolonged expiration, with comparatively few râles, the harsh bronchial breathing, and the general objective symptoms of the case will explain the cause of the pulmonary condition.

The dyspnœa due to a foreign body in the air-passages, whether it be a piece of meat or a false membrane, is quite different from

that of asthma, for in this case the difficulty is commonly in the entrance of air. The onset of the attack is usually sudden, but inspection will show that on inspiration the costal interspaces are greatly drawn in, as is also the epigastrium. There will be practically no signs in the chest which are not evidently due to the efforts at forced breathing, and a history of having had a foregin body in the mouth or of some laryngeal disease will usually be obtainable. Obstruction may, however, be present and this history be absent, in cases in which an abscess has burst into the air-passages from the mediastinum or through the posterior pharyngeal wall. In such a case, however, there would be, in all probability, purulent expectoration.

Laryngeal spasm, producing difficult breathing, causes symptoms precisely like those of foreign body in the larynx, except that in spasm a cough is often constant and is very brassy or ringing. The patient will show by a gesture with his hand that the obstruction is in the larynx, if unable to speak. Such obstruction when seen in children is due to spasmodic croup as a rule, and, if so, probably depends upon one of three causes, namely, laryngeal catarrh, rickets, or digestive disturbance. If in an older person, it is probably due to aneurism pressing on the recurrent laryngeal nerve, to locomotor ataxia, or growths in the mediastinum producing pressure on the nerve-trunks going to the laryngeal muscles. Sometimes great enlargement of the retro-bronchial glands will cause laryngeal spasm, or obstruction by pressure.

Tumors occur in the chest generally as mediastinal growths, and are most commonly sarcomata or lymphadenomata. There will be found, if the growth be large, evidences of its pressure upon the chest-wall such as bulging and dulness on percussion over the swelling. This level of dulness is unaltered by changing the posture, as it would be in pleural effusion. Generally there will be evidence of pressure on the bronchial tubes, which causes dyspnœa, and of pressure on the thoracic vessels, which produces signs of impaired circulation as shown by cyanosis, venous engorgement, and flushing of the skin of the face and neck. Often such growths cause pleural effusions by pressure on the bloodvessels, or produce pulmonary consolidation by causing an exudation into the lung-tissue.

The diseased conditions from which it is necessary we should distinguish mediastinal growths during life are as follows: 1. From aneurism. 2. From abscess. 3. From pleural effusion. And 4.

From chronic pneumonia. There are several subdivisions of these diseases that might be made, but to all intents and purposes these are sufficient. Pericarditis may perhaps be named as the fifth lesion to be thought of.

Aneurism in the thorax is sometimes so extremely difficult of absolute diagnosis that but few rules can be laid down for its differential diagnosis from growths in the mediastinum, for deeply seated aneurism in this region cannot be said to possess any pathognomonic symptoms. The various portions of the aorta in which aneurism occurs make its symptoms different in almost every case, and we are forced to rely more upon general conditions than absolute signs. Thus, if a patient has no direct symptoms of aneurism, and none of those conditions present which we know predispose to such a lesion, such as atheroma of the bloodvessels, due to Bright's disease or any other similar cause, or syphilis, rheumatism, or a history of violent exertion or severe toil, we may with a certain degree of assurance look further for symptoms of mediastinal trouble of another sort. (See "Aneurism" in this chapter.)

Unfortunately, the most common age for aneurism is much the same as that for mediastinal disease, although mediastinal disease seems to occur more frequently in youths than does aneurism, or, in other words, is scattered over a wider range of years. The pain of aneurism is generally considered to be more violent than that of any other thoracic lesion, but there exists reasonable doubt whether the lancinating pain of a growth in this position does not exceed it. This doubt rests on sufficient basis to prevent one using this symptom as an aid in any way to diagnosis. If the aneurismal sac be large enough to give us a wide area of dulness on percussion, as Dr. Graves has stated, there ought to be an expansile movement. Hæmoptysis is not in any way a differential sign, since in the one case it may be due to aneurismal leakage, and in another to ulceration of small bloodvessels by pressure exercised by a tumor, be it aneurismal or malignant, or even benign.

From abscess the diagnosis of mediastinal tumors is much more readily made. In the first place, in abscess we generally have a history of traumatism, or, if the case be one of cold abscess, it is commonly associated with a history of struma. If the abscess be acute, there is generally the history of pain, followed by a chill more or less severe, and fever ; or, if cold, then we frequently have irregular febrile movements, with long-continued anorexia and loss

of flesh. Cold abscess, too, is generally in the posterior mediastinum, while acute abscess generally occurs in the anterior space.

Pulsation may frequently occur, owing to the transmission of the aortic or cardiac impulses, and affords no better diagnostic point here than elsewhere. In some cases, where the theory of aneurism is extremely doubtful and the likelihood of abscess extremely probable, an exploratory needle may be used, either through a hole drilled in the sternum or passed between the ribs; but a careful review of the history of the case should certainly always be made and used as a basis from which to draw conclusions.

By far the greatest difficulty may be experienced when we attempt to diagnosticate between pleural effusion produced by pleurisy and pleural effusion produced by mediastinal disease, provided the case be not seen from the first and the history be obscure. If the effusion be not great, we may be able to discern friction-sounds produced by the rubbing of the tumor against the chest-walls; but if the effusion be large, this sign may not be recognizable. All other methods failing, it would be advisable to tap the chest, and, if the fluid drawn be fibrinous, we know it to be inflammatory; while if it be clear and limpid, or at least thin and not viscid, it is probably due to pressure. This is not, however, a positive sign, since very frequently in cases of asthenic inflammation we have an exudate lacking entirely in the fibrinous constituents.

Tumors of the mediastinum invading the lungs have frequently been mistaken for chronic and even acute pneumonia, passing, as they do, along the larger bronchial tubes and bloodvessels.

Without doubt, in a certain number of cases, either hypostatic pneumonia, or pneumonia due to pressure on the bronchial vessels, develops as the tumor invades the lung, and in such cases it is absolutely impossible to make any diagnosis unless by symptoms of pressure in the mediastinum, or some history pointing to such a result. Walsh has stated that if the lesion be due to a tumor, the affected side will increase in bulk rather than diminish, and that dyspnœa out of proportion to the degree of consolidation points to a mediastinal disorder rather than one confined to the lungs. If the heart be displaced in either direction, the odds point to mediastinal tumor, but the presence or absence of a hæmoptysis, as has just been stated, influences the diagnosis not at all.

The diagnosis of pericarditis from mediastinal lesions is much more readily made. The history of sudden præcordial pain and the

limited area of dulness on percussion aid us very materially in deciding as to what the disorder is, while the description of the onset of the attack, with a few pointed questions as to systemic taints, etc., may do much to unravel the mystery. The distention of the pericardial sac from effusion may give us a regular outline on percussion, while the dulness of mediastinal disease may be irregular and varying.

Heart-sounds and Signs.

On attempting to study the heart-sounds we usually auscult the neighborhood of the apex-beat and expect to find, if the heart be healthy, two sounds, occurring one immediately after the other, which resemble the sounds of the words "lub dup;" the "lub" being the so-called first sound of the heart produced by the contraction of the heart muscle and the tense valves, and the "dup" being caused by the slapping to of the aortic valves. After listening in this region we next place the ear over the second right costal cartilage in order to come as near as possible to the point of origin of the second sound produced by the aortic valves. If the heart is normal, we find only these sounds, "lub dup," and nothing else. If it is feeble from exhausting disease, from fainting, or by reason of fatty degeneration, we find that the sound "lub" is feeble, and the "dup" sound is also feeble, because the valves do not slap back into place with as much force as is normal. If, on the other hand, the heart is hypertrophied or stimulated, we find these sounds accentuated, and it is of importance to remember that marked accentuation of the aortic second sound, showing forcible closure of the aortic valves, indicates a condition of high arterial pressure, often the result of vascular spasm arising from chronic contracted kidney. On the other hand, if the pulmonary second sound at the second left intercostal space is accentuated, it indicates an increase in pulmonary pressure due to impediment to the flow of blood in the lungs. It is markedly accentuated in both mitral obstruction and regurgitation and in some cases of pneumonia.

The sounds produced at the various orifices of the heart are heard best at the following points (Fig. 125): The mitral valve is heard best at the apex-beat; the aortic valve at the second right costal cartilage, the tricuspid valve over the sternum on a line drawn from the third left intercostal space to the fifth right costal cartilage,

and the pulmonary valve at the second left intercostal space. All the heart-sounds may be reduplicated in health and in disease as the result of contraction in an unequal manner of the papillary muscles. If disease of the valves be present, we are apt to find reduplication of the second sound in cases of mitral stenosis and lung disease producing an abnormally high tension in the pulmonary circulation. Such reduplication is also seen in some individuals suffering from aortic stenosis.

Fig. 125.

Showing the areas in which the various heart-sounds are best heard in health. A is the area for the aortic valve; P, that for the pulmonary valve, T, for the tricuspid valve; and M, for the mitral valve.

Supposing that on listening to the heart in the mitral area there is heard in place of the normal sounds ("lub dup"), or with them, a murmur, what does it mean? It means that, friction-sounds being excluded, either valvular disease of the heart, aneurism of the aorta, or marked anæmia is present. Particularly is the anæmic murmur apt to be heard in case of feeble children suffering from chorea, and it will generally be found most marked at the left margin of the sternum.[1]

Having found that there is a murmur, and, from the absence of anæmia, that it is due to organic cardiac disease, it is now necessary to determine at what orifice of the heart it is produced, and the rule is to be remembered that a murmur is always heard loudest at about its point of origin. We therefore place the ear over the aortic cartilage (second right). If the murmur be mitral in origin, it will not be heard at this place, unless it be so loud as to be transmitted. If it is aortic in origin, it will be louder here than at the apex. If it is tricuspid, it will be loudest in the tricuspid area; if pulmonary, loudest at the pulmonary area. As murmurs at the tricuspid and pulmonary valve are rare, we nearly always have to deal with aortic

[1] It must not be forgotten that murmurs due to endocarditis also are frequently found in choreic children.

or mitral murmurs or both. In this way, therefore, we can determine the origin of the murmur, and that it is a mitral or an aortic murmur. Let us suppose that it is mitral. We must determine whether it is that of mitral regurgitation or obstruction, or, as they are also called, incompetence and stenosis. The probabilities are that it is the regurgitant murmur, because this lesion is by far the most common murmur heard in the heart; and if to this probability we add the fact that it is transmitted well into the axilla, and even heard at the angle of the scapula, our diagnosis is greatly aided, for this is the area of transmission of the murmur of mitral regurgitation. The most important diagnostic point, however, is the discovery that the murmur occurs simultaneously with the first sound of the heart, or with systole—that is, with the apex-beat or the carotid pulse. If it does, and the other signs of mitral disease are present, it is almost certainly one of mitral regurgitation. This murmur occurs with the first sound, or systole, because the ventricle in contracting drives most of the blood in the normal direction into the aorta, and also forces some of it back through the auriculo-ventricular orifice into the auricle, causing a regurgitant murmur. There will be found very often in such cases a very marked accentuation of the second sound at the pulmonary orifice. The area of greatest intensity of the mitral regurgitant murmur is shown in Fig. 126.

Fig. 126.

Showing at x the apex-beat where the murmurs of mitral regurgitation and obstruction can be best heard. The arrow pointing to the axilla indicates the direction in which the regurgitant murmur is transmitted, and the arrow pointing to the sternum the direction of transmission of the obstructive murmur.

In adults inspection and palpation will rarely reveal much of a thrill over the præcordium in mitral regurgitation, but in children this thrill is rarely absent and is usually well marked. Percussion will show that the area of cardiac dulness (see earlier part of this chapter)

is broadened, extending beyond the right edge of the sternum and to the left of the mammillary line. (See Fig. 120.)

If, on the other hand, it is found that the murmur does not occur with systole, but just before it, is not transmitted into the axilla, but to the right, over to the midsternal line, it is probably that of mitral stenosis—that is, the presystolic mitral murmur (see Figs. 126 and 127). This murmur can often be exaggerated by placing the patient

Fig. 127.

MO shows area of greatest intensity of a mitral obstructive murmur. TR shows area of greatest intensity of a tricuspid regurgitant murmur.

in a prone position, and occurs before systole, or the first sound, because it is made by the blood passing through an obstructed auriculo-ventricular orifice, and, as the ventricle does not contract (systole) till it is filled, the murmur must be made while it is filling, and so is presystolic in time. Palpation of the præcordium in such a case will usually reveal a marked thrill in the fourth or fifth interspaces. If the compensation of the heart in a case of mitral stenosis is broken, these signs are accompanied by a very irregular action of the heart, the first sound becoming accentuated and the murmur disappearing. In some cases what is called a "gallop rhythm" develops, the heart-

sounds being reduplicated in such a manner as to make a galloping sound.

If, however, we have found the murmur to be aortic, we have now to exclude aneurism and then determine whether it is that of aortic regurgitation or obstruction. The characteristic symptoms of aortic aneurism are a "bruit" or angry murmur, systolic in point of time, and heard best in the area between the third rib and the clavicle on the right side, accompanied by pulsation, which is often

FIG. 128.

Case of aortic and innominate aneurism, with erosion of the clavicle and ribs, from the author's wards in the Jefferson Hospital. This case is of extraordinary interest because this picture was taken thirty-five months after the author's friend, Dr. D. D. Stewart, had caused an arrest of the growth of the aneurism by electrolysis. The man finally entered the Jefferson Medical College Hospital because the subjective symptoms of aneurism were returning, and a spot of softening and pulsation was to be felt in the lower right-hand border and at the edge next the xiphoid cartilage. This same patient is seen laterally in Fig. 109.

expansile; by an angry thrill; by bulging of the chest-wall (Figs. 109 and 128) if the disease is advanced and presses forward; by glossiness of the skin over the bulging; by unilateral sweating of the face and neck from pressure on the cervical sympathetic; by inequality of the pupils from the same cause; and by inequality of the radial pulses, owing to the delay caused the blood in entering the vessels of the arm on one side. Again, we usually have a very considerable degree of dyspnœa, and sometimes dysphagia from

pressure on the œsophagus. There may also be history of syphilis and of severe strain or injury in many cases.

Although these are the general symptoms of aortic aneurism, there are others which depend upon the seat of the aneurism and which materially modify the points so far named in diagnosis. Let us suppose that a patient presents himself with great engorgement of the vessels of the head and neck and arm of the right side, with perhaps œdema of the arm. The heart may be pushed downward and to the left and the voice may be lost or partially impaired by pressure on the recurrent laryngeal nerve of the right side. Generally such symptoms will be due to an aneurism of the greater curvature of the ascending aorta, although they may be due to a tumor in the anterior or middle mediastinum, but the expansile pulsation, the bruit, and the history of the case will often make the differentiation possible. Such a growth will probably cause bulging of the second or third interspace on the right side. Again, let us suppose that the chief symptoms manifested by the patient are a brassy, ringing, and constant cough, with difficulty in swallowing, and an examination of the vocal cords shows that the left cord is paralyzed rather than the right, thereby causing hoarseness, and there is more or less bulging and distention of the innominate or left carotid arteries, while the radial pulse is delayed, then we may consider that the aneurism is probably of the transverse portion of the arch. Still further, if an irregular action of the heart, with great pain in the back, is found, in addition to the ordinary signs of aneurism, it is possible that the aneurism may be in the descending aorta and be eroding the vertebra. If the aneurism be of the first two forms named, there may be found, on percussion, dulness over the swelling or over the area of expansile thrill and bruit. Thus the aneurisms of the ascending part of the arch which project forward and to the right cause dulness on the right side of the sternum, while those of the transverse part of the arch cause dulness on percussion on the left side or in the middle line. On the other hand, the lesion of the descending aorta often causes dulness and bulging between the vertebral column and the scapula on the left side.

There are other symptoms connected with aneurism which should not be overlooked. The first of these is "tracheal tugging," a sign which is found in some cases of aneurism. The patient being in the erect position the fingers of the physician grasp the cricoid cartilage and gentle upward traction is produced. If aneurism is pres-

ent, a distinct tug will be felt with each beat of the heart. Another sign mentioned by Osler is the loss of pulsation in the peripheral vessels, the result of the loss of the heart's impulse in the aneurismal sac.

If the symptoms of aortic aneurism are excluded, we proceed to determine the question as to whether the murmur is that of aortic stenosis or obstruction or incompetence or regurgitation. Aortic obstruction is the more common lesion of the two. This murmur occurs with the systole of the ventricles, or the carotid pulse or apex-beat; it is harsh as a rule, and is transmitted up into the carotids and it may be into other arteries of less importance. (See Fig. 129.) It is produced by the contraction of the ventricle driving the blood through a narrowed or roughened aortic orifice. Considerable hypertrophy of the left ventricle is usually present, and the apex-beat is strong and forcible.

FIG. 129

Showing the area of greatest intensity and the direction of transmission into subclavian and carotid artery of the aortic obstructive murmur.

FIG. 130.

Showing the area in which the murmur of aortic regurgitation can be most clearly heard.

If, on the other hand, the murmur occurs after the systole or apex-beat, and is aortic, the murmur is that of aortic regurgitation, and is called the diastolic aortic murmur. It is heard loudest at the aortic cartilage, but is transmitted down along the sternum very

clearly and into the left ventricle, so that it is heard at the apex. (See Fig. 130.) In this condition we have usually marked dilatation of the heart with hypertrophy (the so-called "ox-heart"), and a peculiar trip-hammer pulse (see chapter on Pulse), sometimes called the "water-hammer" or Corrigan pulse. This murmur is due to incompetence of the aortic valve, which allows the blood to regurgitate into the heart after it is driven out into the aorta.

If the examination has shown that the tricuspid valve is diseased, it is to be remembered that in the vast majority of cases the murmur is due to tricuspid regurgitation, for tricuspid stenosis is an exceedingly rare condition. The time of the murmur of the tricuspid lesion is identical with that of the mitral regurgitant (systolic), because this valve is the counterpart in the right side of the heart of the mitral valve.

Actual disease of the pulmonary valves is exceedingly rare, and the regurgitant form of lesion is almost never met with. The murmurs sometimes heard, and the thrills sometimes felt, in this area are generally due to anæmia, the puerperal state, or some neurosis; or to congenital narrowing of the pulmonary artery; or to compression of the vessel by the heart. If the last two causes are present, the ventricular septum is usually deficient and cyanosis is noticeable.

In the diagnosis of all murmurs in the heart we must remember that several valves may be diseased, producing associated murmurs. Some discussion as to the relative frequency of these associations has arisen, but the results of H. J. Smith in the London hospitals are usually accepted as correct. His results are as follows, in the order of frequency and association:

1. Aortic regurgitation and stenosis and mitral regurgitation.
2. Mitral stenosis and regurgitation.
3. Aortic stenosis and mitral regurgitation.
4. Aortic regurgitation and mitral stenosis.
5. Aortic regurgitation and stenosis.
6. Aortic regurgitation and stenosis: mitral stenosis and regurgitation.
7. Mitral regurgitation and tricuspid regurgitation.
8. Aortic regurgitation and stenosis; mitral regurgitation; tricuspid regurgitation.
9. Mitral stenosis and regurgitation; tricuspid regurgitation.
10. Aortic stenosis; mitral stenosis and regurgitation.

11. Aortic regurgitation ; mitral stenosis and regurgitation.

12. Aortic stenosis ; mitral regurgitation ; tricuspid regurgitation.

13. Aortic regurgitation and stenosis ; mitral regurgitation ; pulmonary regurgitation.

14. Aortic stenosis and regurgitation ; mitral stenosis.

15. Aortic regurgitation ; mitral stenosis.

16. Aortic regurgitation ; mitral regurgitation ; tricuspid regurgitation.

17. Mitral stenosis ; tricuspid regurgitation.

18. Aortic stenosis ; mitral stenosis and regurgitation ; tricuspid regurgitation.

19. Aortic stenosis ; mitral stenosis.

20. Aortic regurgitation and stenosis ; mitral stenosis and tricuspid regurgitation.

21. Aortic regurgitation ; mitral stenosis and regurgitation ; tricuspid regurgitation.

22. Aortic regurgitation and stenosis ; mitral stenosis and regurgitation ; tricuspid regurgitation.

23. Aortic regurgitation and stenosis ; mitral stenosis and regurgitation ; tricuspid stenosis and regurgitation.

24. Aortic stenosis ; pulmonary stenosis.

25. Aortic stenosis ; mitral stenosis and regurgitation ; tricuspid stenosis and regurgitation.

26. Mitral stenosis and tricuspid stenosis.

The relative gravity of heart-lesions is, according to Walsh, as follows, the least dangerous being placed last and the most dangerous first :

Tricuspid regurgitation.
Mitral obstruction and regurgitation.
Aortic regurgitation.
Pulmonary obstruction.
Aortic obstruction.

The general symptoms, subjective or objective, which a patient suffering from the various forms of valvular lesion presents, in some instances, have not been spoken of up to this point, because it is to be distinctly understood that murmurs produced by any form of valvular lesion may exist with great intensity without there being any systemic disturbance or the patient being conscious of their presence. On the other hand, the murmur may be so faint as to be almost indistinguishable, and yet the general symptoms of heart disease be

very marked. This is because the development of general symptoms depends entirely upon the question of compensation by hypertrophy. If there is a leak in a valve or a constriction of an orifice, this leak or obstruction must be overcome by compensatory hypertrophy of the heart-muscle. If the heart-muscle can make up for the regurgitation or obstruction by increased effort, the circulation is unimpaired; but if it cannot do so, we have developed more or less rapidly, according to the lesion present and the condition of the heart-muscle, characteristic symptoms. Let us suppose that the valvular lesion is that of mitral regurgitation with failure of compensation. The first and one of the most prominent symptoms, is shortness of breath on exertion; the lips and ears do not possess their normal red hue, but are a little bluish; and if the congestion of the auricle and pulmonary veins is great, bronchitis may be constant or attacks of hæmoptysis may develop. Palpitation of the heart will also be complained of; and if the patient has developed the lesion in early life, the finger-tips are apt to be clubbed. If the rupture or failure of compensation is more complete, all these symptoms become more marked, and the shortness of breath, even when lying down, becomes most distressing; indeed, the patient may be comfortable only when sitting up. Dropsy of the lower extremities now comes on and the liver becomes enlarged from portal congestion, while the urine becomes albuminous, not from any true renal lesion, but as the result of its engorgement with blood.

The general symptoms of mitral obstruction are identical with those just described.

The general symptoms of aortic obstruction are also much like those described as resulting from mitral regurgitation, but in addition there are apt to be present, early in the process of failing compensation, some lightness of the head, dizziness or vertigo, or faintness, owing to a deficient blood-supply to the brain. Very commonly, too, it will be found that in association with the aortic stenosis there also exists mitral regurgitation, which speedily produces in its turn well-marked pulmonary symptoms. Dropsy is very rarely seen in patients with aortic stenosis. On the contrary, they present, as a rule, the lean and poorly nourished appearance so often found in the adult, well-advanced in years, with atheromatous tendencies in his vessels.

The association of ruptured compensation with aortic regurgitation presents more typical general systemic symptoms than any of

the ordinary valvular lesions of the heart. In addition to headache, vertigo, and a tendency to syncope, associated with palpitation and a sense of cardiac oppression, we often have a great deal of cardiac pain, of a dull, aching character in rare instances, but more often intensely sharp and lancinating, often darting down the left arm, particularly at night. The dyspnœa is often extreme, the patient suffering from terrible attacks of shortness of breath and often sitting day and night in a chair with his head resting on the back of a chair placed in front of him. As time goes on the constant struggling for breath exhausts him and he falls asleep, only to awake in a few moments gasping to get air. Long before any of these grave symptoms arise we may, however, find a number of interesting signs of this heart-lesion, chief among which is the "water-hammer" or "trip-hammer" or "Corrigan pulse," the throbbing carotid arteries, and capillary pulsation in the skin and mucous membrane is to be seen. This is best developed by drawing the thumb-nail sharply across the forehead, thereby causing a red mark which can be seen paling and flushing with each beat of the heart, or by pressing a glass slide on the inner part of the lower lip, when the same capillary pulsation will be found. Ophthalmoscopic examination will often reveal pulsation of the retinal arteries.

Beyond valvular lesions, producing heart-symptoms, we have a number of other causes which seriously disturb the action of the heart and the general circulatory condition. The first of these is dilatation of the heart. Let us suppose that a man presents himself with a history of shortness of breath on exertion so great that his activities are greatly reduced and his usefulness impaired. He gives a history that he has been well until he had made some extraordinary exertion, generally of a prolonged character, rather than a brief and sudden effort, which would perhaps cause aneurism. Since that time his symptoms of heart-failure have been marked. He may perhaps have attacks of syncope. Examination of his heart reveals on inspection a diffuse thrill in the region of the apex, but this thrill is too feeble to be felt, though well marked to the eye if his chest is thin. Percussion shows that the area of cardiac dulness is increased vertically and laterally, and auscultation will discover feeble heart-sounds; and if the dilatation of the muscular portion of the heart is associated with dilatation of the orifices, a murmur may be present, most commonly that of mitral regurgitation. Sometimes tricuspid regurgitation is also found. The first sound before it becomes very

feeble may be short and flapping like the ordinary second sound. Marked arhythmia of the heart is often present.

Again, we have hypertrophy of the heart occurring in persons without valvular lesions, sometimes as the result of excessive and severe toil. It is seen most commonly by the author in the chests of medical students, who, during their holidays, devote their time to severe athletic sports, or to too much manual labor, and who, on leading sedentary lives in the winter, develop irregular cardiac action, palpitation, and some shortness of breath. Examination of the præcordium in such cases shows a forcible impulse of the apex of the heart against the chest-wall, some bulging of the chest-wall if the hypertrophy be very great, and no murmurs, but in their place heart-sounds very much louder than normal. Palpation shows the apex-beat to be lower than normal, and on percussion an increase in the area of cardiac dulness is also found.

Again, let us suppose that a patient presents himself with a very rapidly beating heart, he tells us that his skin is alternately red and pale, and a careful examination of the heart fails to reveal any murmurs or organic abnormality. There are considerable shortness of breath on exertion and marked palpitation and arhythmia. Such a case may be suffering from a condition in which there is some deficient action of the pneumogastric nerve, whereby the heart is not properly controlled, or the irregular cardiac action may be due to sudden vasomotor relaxations and spasms, which by dilating or closing the blood-paths remove the normal arterial resistance or make it excessive. This is a condition seen in association with some neuroses and very commonly seen in persons who use tobacco to excess. The physical signs of the so-called "tobacco-heart" are indeed chiefly those of arhythmia due to pneumogastric disorder.

Rarely because of irritation of the vagus nerves or centres a state of bradycardia develops, in which the heart beats very slowly, perhaps only thirty or even as slowly as twelve times a minute. Bradycardia or great slowness of the heart may not only be due to a neurosis of the vagi, but to typhoid fever or other infectious diseases. It is also seen in jaundice.

One of the most common causes of tachycardia, or rapid heart, is exophthalmic goitre, in which condition we have not only exophthalmus and enlargement of the thyroid gland, but, in addition to the tachycardia, a marked thrill over the carotid arteries, in which

vessels a purring murmur of considerable intensity can also be heard. The patient often suffers from considerable nervous excitement or mental depression. It is an interesting fact that in this disease the electrical resistance of the body is often diminished.

An exceedingly irregular arhythmical action of the heart coming on in the course of acute infectious disease, or in any state productive of sepsis, points to the possibility of the patient having an embolism or thrombus of one of the coronary arteries. If the vessel is suddenly plugged, death speedily occurs; but if the process is gradual, an anæmic necrosis or white infarct is produced.

Sudden attacks of cardiac feebleness sometimes come on as cardiac crises in glosso-labio-pharyngeal paralysis and in locomotor ataxia.

Before discussing the signs of so-called fatty heart we must decide what is meant by this term. True fatty heart—that is, the condition of the heart in which this organ has undergone true fatty degeneration—has no pathognomonic signs, so far as the heart itself is concerned. In these instances we base our diagnosis upon the presence of fatty degeneration of the more superficial organs, such as the arcus senilis in the eye, the presence of atheromatous blood-vessels, the feeble heart-sounds at all times, and the evident feebleness of the heart on exertion. The history of poisoning by any one of the poisons causing fatty degeneration is also to be sought after in some cases. Marked fatty degeneration is often present in cases of pernicious anæmia. It is not possible to make a differential diagnosis from the physical signs between fatty and fibroid heart.

Another state quite distinct from true fatty heart, but with somewhat similar symptoms, is seen in cases in which an excessive amount of fat has been deposited around the heart as well as in or around the other organs of the body. Here there is little or nothing the matter with the heart-muscle, except that it is overloaded with a weight of fat.

Where a man shows signs of general degenerative changes, has a feeble heart, some dyspnœa, and perhaps some œdema of the lower extremities, we may conclude that he has, unless valvular disease is discovered, degenerative myocarditis. Such cases make up the greater number of sudden deaths, called popularly "death by sudden cardiac failure."

Finally, let us suppose that a young child is seen who is, and has been since birth, more or less cyanotic, according as to whether it is quiet or moving and varying its posture. In all probability

such a child is the subject of congenital malformation of the heart. The following rules, laid down by Hochsinger, may be used for their diagnosis :

1. In childhood loud, rough, musical heart-murmurs, with normal or only slight increase in the heart-dulness, occur only in congenital heart-disease. The acquired defects with loud heart-murmurs in young children are almost always associated with great increase in the heart-dulness.

2. In young children heart-murmurs, with great increase in the cardiac dulness and feeble apex-beat, suggest congenital changes. The increased dulness is chiefly of the right heart, whereas the left is only slightly altered. On the other hand, in the acquired endocarditis in children, the left heart is chiefly affected and the apex-beat is visible ; the dilatation of the right heart comes late and does not materially change the increased strength of the apex-beat.

3. The entire absence of murmurs at the apex, with their evident presence in the region of the auricles and over the pulmonary orifice, is always an important element in differential diagnosis, and points rather to septum defect or pulmonary stenosis than to endocarditis.

4. An abnormally weak second pulmonic sound associated with a distinct systolic murmur is a symptom which, in early childhood, is only to be explained by the assumption of a congenital pulmonary stenosis, and possesses, therefore, an importance from a point of differential diagnosis which is not to be underestimated.

5. Absence of a palpable thrill, despite loud murmurs which are heard over the whole præcordial region, is rare, except with cogenital defects in the septum, and it speaks therefore against an acquired cardiac affection.

6. Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle, are very important, for the diagnosis of a persistence of the ductus Botalli, and cannot be explained by the assumption of an endocarditis of the aortic valve.

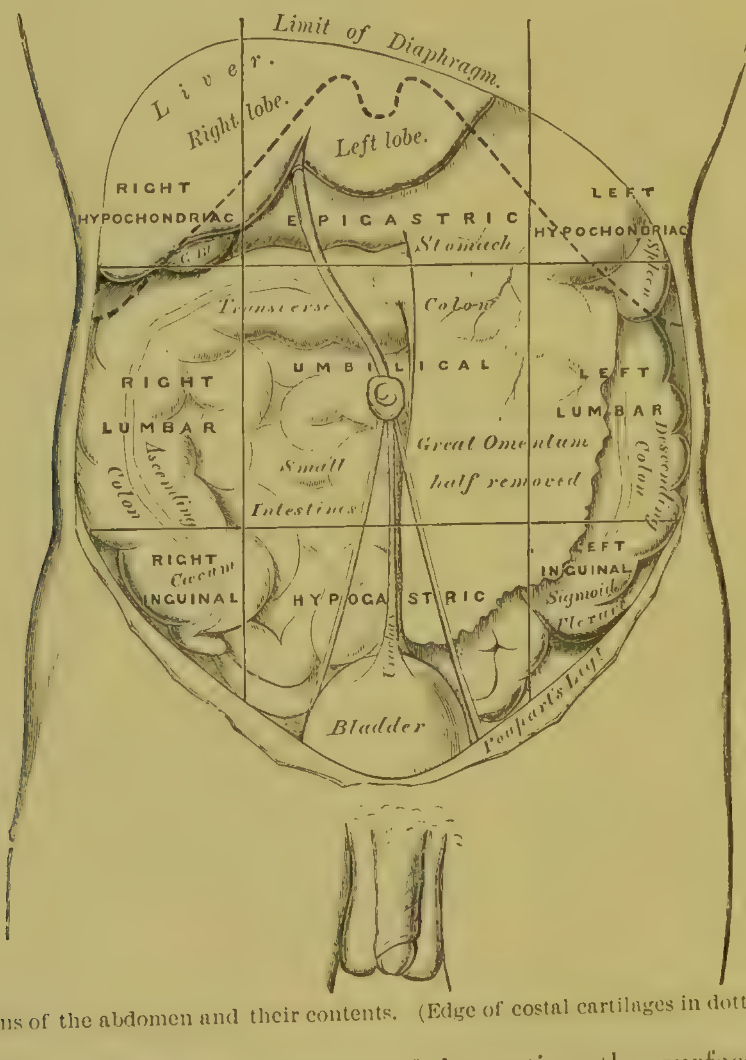
CHAPTER IX.

THE ABDOMEN AND THE ABDOMINAL VISCERA.

The surface of the abdomen — Changes in the appearance and shape of the abdominal wall—The signs and symptoms of disease of the abdominal organs.

THE condition of the abdominal surface and abdominal contents is best studied by means of inspection, palpation, percussion, and

FIG. 131.



The regions of the abdomen and their contents. (Edge of costal cartilages in dotted outline.)

auscultation. For the purposes of inspection the surface of the abdomen has been arbitrarily divided by diagnosticians into a num-

ber of spaces, which are best shown in the accompanying figure (Fig. 131), and which get their names from the regions in which they are located, or from the organ immediately underneath the abdominal wall. By means of these arbitrary outlines we can readily describe the exact spot in which a physical sign or symptom is found.

The table which is appended, from Gray's *Anatomy*, clearly shows the viscera to be found under each of the areas named :

Right Hypochondriac. The right lobe of the liver and the gall-bladder, hepatic flexure of the colon, and part of the right kidney.	*Epigastric Region.* The pyloric end of the stomach, left lobe of the liver, and lobulus Spigelii, the pancreas, the duodenum, parts of the kidneys and the supra-renal capsules.	*Left Hypochondriac.* The splenic end of the stomach, the spleen and extremity of the pancreas, the splenic flexure of the colon, and part of the left kidney.
Right Lumbar. Ascending colon, part of the right kidney, and some convolutions of the small intestine.	*Umbilical Region.* The transverse colon, part of the great omentum and mesentery, transverse part of the duodenum, and some convolutions of the jejunum and ileum, part of both kidneys.	*Left Lumbar.* Descending colon, part of the omentum, part of the left kidney, and some convolutions of the small intestine.
Right Inguinal (Iliac). The cæcum, appendix cæci.	*Hypogastric Region.* Convolutions of the small intestine, the bladder in children, and in adults if distended, and the uterus during pregnancy.	*Left Inguinal (Iliac).* Sigmoid flexure of the colon.

INSPECTION. The general abdominal wall is pushed outward or protruded by many perfectly normal causes, such as an unusual amount of fat in the omentum, pregnancy, and an accumulation of liquid and food in the stomach after a heavy meal. It is also convex to an abnormal degree in cases in which ascites is present, when the stomach and bowels are over-distended with gas (tympanites), and when any of the organs found in the peritoneal cavity are the seat of swellings or tumors of large size. In children a protruding pot-belly, "the frog-belly" of the French, is seen in cases of scrofula or tuberculosis of the mesenteric glands, and in those cases which suffer from chronic gastro-intestinal catarrh. It is claimed in a recently published paper by a French clinician that the intestinal canal is not only dilated but of greater length than is normal in these cases. If, on the other hand, the belly-wall is retracted, concave, or "scaphoid" as it is sometimes called, we look for the cause in abstinence from food, or remember the possibility that excessive vomiting or purging may have emptied the gastro-intestinal tract of

its usual contents. Thus excessive summer diarrhœa may produce such a result. We also find a retracted belly-wall in nearly all cases of advanced wasting diseases, such as carcinoma or tuberculosis of the lungs; and if the retraction is associated with muscular rigidity of the belly-wall and pain, we suspect the early stages of peritonitis or the presence of some acutely painful affection, such as renal or hepatic colic or lead colic. Marked concavity and retraction of the belly-wall are also seen sometimes in cases of tubercular meningitis.

The abdomen is distended very greatly by gas in many cases of peritonitis, typhoid fever, and flatulent colic, and the anterior surface of the belly will be found to give a high-pitched tympanitic note on percussion. We separate, diagnostically, the swollen abdomen due to wind from that due to ascites by the fact that in the latter condition the epigastrium is moderately flat, while in the case of tympanites it is more protruding. Again, in ascites the greatest bulging is often seen in the flanks, or, if the patient sits or stands erect, the hypogastric region bulges from the change in the position of the fluid. If the fluid be due to a moderate-sized ovarian cyst, this variation will not occur, as the cyst is not readily movable. If the ovarian tumor be large, the differential diagnosis may be most difficult and almost impossible, except by the history or by examining the liquid withdrawn by tapping. The following table from Brown's *Diagnosis* aids in this diagnosis:

Ascites.	*Ovarian Cyst.*
I. History.	I. History.
No history of lateral development.	Tumor develops from one iliac fossa.
II. Inspection.	II. Inspection.
When patient lies on the back there is bulging at the flanks. If the ascites is considerable, the umbilicus is pressed outward.	The greatest swelling is anterior, not in the flanks. Sometimes one side of the abdomen is more prominent than the other.
III. Percussion.	III. Percussion.
On percussion there is dulness in the flanks, and a clear note over the centre of the abdomen.	The dulness is central, the intestines giving a clear note at the sides.
Changes of position alter the lines of dulness in the manner already described.	Change of position does not alter the lines of dulness.
IV. Aspiration.	IV. Aspiration.
Ascitic fluid presents the following characters:	Ovarian fluid has the following characters:
1. Specific gravity 1.010 to 1.015.	1. Specific gravity 1.018 to 1.024.
2. Light straw color.	2. Amber colored; often syrupy.
3. Coagulates spontaneously when exposed to the air.	3. Seldom or never coagulates spontaneously.
4. Does not contain paralbumin.	4. Contains paralbumin.

In cases of ascites due to free liquid in the abdominal cavity percussion will elicit flatness over the flanks and resonance only when the intestines containing gas are floated up against the anterior belly-wall in front of the effusion. Palpation will also reveal fluctuation in ascites, but none in tympanitic distention. As the result of gradually increasing intra abdominal pressure the floating ribs become pressed outward, the apex-beat of the heart is often displaced

FIG. 132.

Dotted line shows area of cancerous liver extending far beyond its normal area. Over the entire surface of this mass could be felt hard nodular masses. (From the author's wards, Jefferson Medical College Hospital.)

upward and outward, and the umbilicus becomes protruded instead of retracted. The skin of the belly-wall becomes thin and shining, and the recti muscles becoming separated render the peristaltic movements of the bowels readily felt through the intervening skin. Very often there is developed in cases of ascites, particularly when that condition arises from hepatic cirrhosis, a more or less well-defined bunch of veins on the anterior belly-wall, which is sometimes called the *Caput Medusæ*, as the result of an attempt at

collateral circulation to compensate for the obstructed flow caused by the changes in the liver. Sometimes a mediastinal growth will cause a somewhat similar development. When the pressure is lower down than the liver the veins of the lower part of the abdomen (hypogastrium) will be found distended.

FIG. 133.

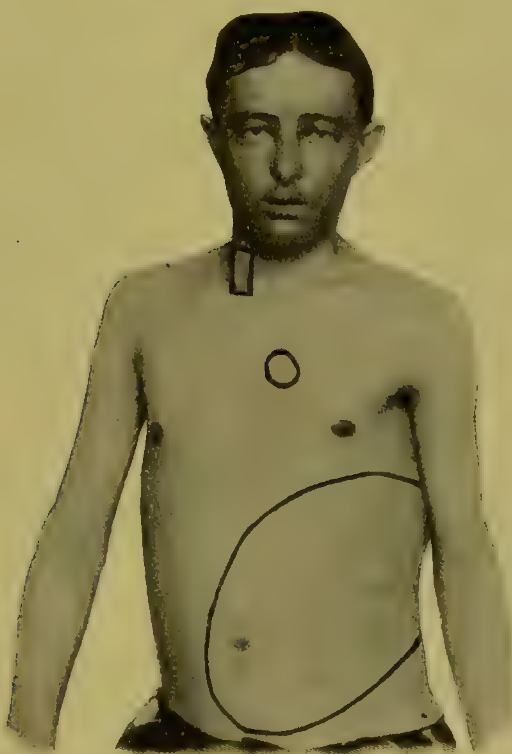
A case of chronic enlargement of the spleen following typhoid fever. The dark line shows the margin of the organ on palpation, while the retraction in the line and the dotted line indicate the position of the splenic notch. (From the author's wards in the Jefferson Medical College Hospital.)

Localized bulging of the abdominal walls, chiefly on the right side, is found in cases in which the liver is enlarged by hypertrophic cirrhosis, or by cancer or other morbid growth, such as gumma or sarcoma, and by abscess. The swelling, if its origin be in the liver, will arise under the floating ribs on the right side, and will extend downward and forward toward the umbilical area. If the enlargement be great, it will extend far below the umbilicus and across the umbilical area to the opposite side of the abdomen. (Fig. 132.) In

enlargement of the spleen similar signs, springing from the floating ribs well over to the left side, may be developed (see Figs. 133 and 134), and large cystic kidney on either side may cause abdominal bulging, particularly if it be floating.

Marked swelling of the epigastrium indicates distention of the stomach by gas, by food, or that this organ is the seat of morbid growth. Sometimes a similar distention results from enlargement of the posterior mediastinal and retro-peritoneal glands. Again,

FIG. 134.



A case of profound anæmia with great enlargement of the spleen, as shown in the large outlined area. The smaller outlines indicate the areas of anæmic murmurs near the base of the heart and in the carotid artery.

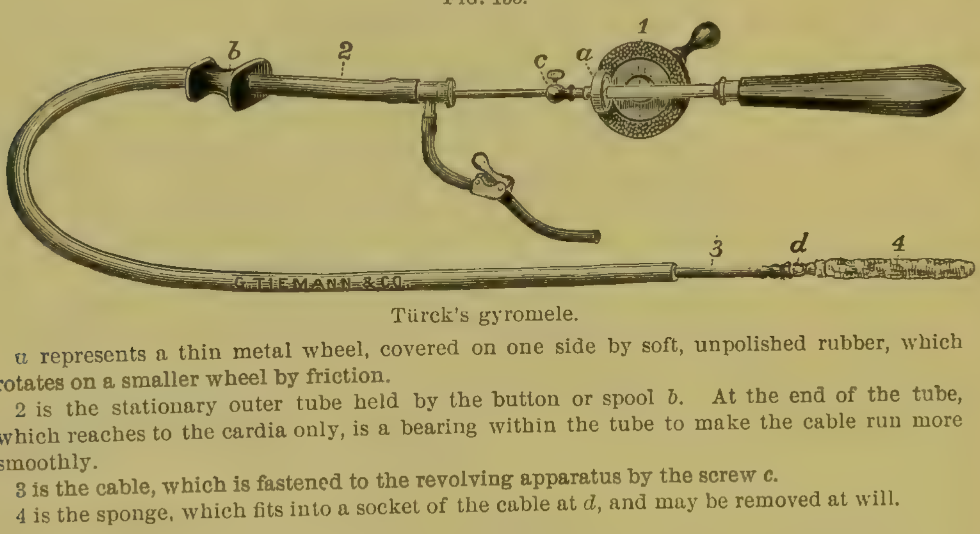
distention of the epigastrium is apt to be caused by enlargement of the left lobe of the liver. In ovarian tumors the growth often gradually distends the entire belly equally; but, as already stated, the history is usually that of swelling, low down, and of its being chiefly unilateral at first.

It should be remembered that the discovery of a pyriform swelling in the hypogastrium in a female may possibly be due to a pregnant uterus, or in a man to retention of urine, with consequent distention of the bladder. Cases of dilatation of the stomach often show very

great bulging of the umbilical area of the abdominal wall when that viscus is distended by food and gas. (See Percussion in this chapter.)

We discover the condition of the stomach as to its size and shape by means of washing it clean with the stomach-tube and then filling it with a known quantity of water, which can be siphoned out and measured. Or we can use the so-called gastrodiaphane of Einhorn, which consists of a small electric lamp, protected by strong glass, and attached to a rubber tube which contains the necessary wiring for the electric current, and which is swallowed just as is the ordinary stomach-tube. The stomach having been thoroughly cleansed by lavage is then filled with pure water and the lamp swallowed. If the patient be moderately thin and the inspection is made in a dark room, the outline of the lighted stomach can be seen through the abdominal wall, and some idea of its dimensions obtained. Normally, the greater part of the stomach will be found to the right of the middle line and only about one to two inches above the umbilicus. (See Percussion.)

FIG. 135.



Türck's gyromele.

a represents a thin metal wheel, covered on one side by soft, unpolished rubber, which rotates on a smaller wheel by friction.

2 is the stationary outer tube held by the button or spool *b*. At the end of the tube, which reaches to the cardia only, is a bearing within the tube to make the cable run more smoothly.

3 is the cable, which is fastened to the revolving apparatus by the screw *c*.

4 is the sponge, which fits into a socket of the cable at *d*, and may be removed at will.

Another useful means of diagnosing dilatation of the stomach is by means of the "gyromele" of Türck, which in its improved form consists of four parts, namely: A revolving apparatus; a stationary outer tube; a cable covered with tight-fitting rubber tube; and a sponge-spiral attached to the cable.

Türck says: "To show the outline of the greater curvature, a tube containing a cable with a sponge at its extremity is intro-

duced into the stomach. An apparatus for producing revolutions is attached to the outer end of the cable. The cable is passed onward and it glides along the great curvature, plainly showing the outline of the stomach. Moving onward, the cable passes upward toward the pylorus, and then turns and passes along the lesser curvature. When rapid revolutions are produced the sponge and cable can be felt in their respective situations. To determine the distensibility of the stomach, cables of different degrees of flexibility are used. A very flexible cable (No. 1) is used first. It is introduced until it meets with resistance at the lesser curvature, and its length is noted. At the same time the revolving sponge is examined by palpation through the abdominal wall. A stiffer cable (No. 2) is then used, and pushed onward until it meets with resistance at the lesser curvature, and its length is noted. The lengths are compared, and their difference shows the degree of distensibility. The degree of distention also is found by palpation through the abdominal wall."

(For the symptoms of gastric dilatation, see the chapter on Vomiting, and that part of this chapter on the Diagnosis of Gastric Carcinoma with Dilatation.)

In inspecting abdominal swellings the physician should watch to see if they move up and down with respiration. If they do, they are probably connected with the diaphragm and depend upon disease of the liver and spleen, as tumors of the pancreas, stomach, and kidney are usually not attached to the diaphragm, and therefore generally do not move. Inspection of the abdominal wall will also show possible venereal infection if the glands in the groin are enlarged, or if in suppurating they have left puckered scars. If silvery lines extend across the belly, they may indicate pregnancy past or present, or any state of the abdominal cavity causing great stretching of the skin. Great bulging in the neighborhood of the umbilicus will naturally suggest umbilical hernia, and swelling in the groin, not due to pus, inguinal hernia, or perhaps an appendicular abscess.

Palpation and Percussion. More important than any other external method of studying the condition of the abdominal contents is the use of gentle *palpation*, the fingers being gradually worked down into the abdominal cavity in such a way as not to cause pain or excite the muscles of the abdominal wall to resistance. The hand should always be carefully warmed before palpation is attempted, and the object of the examiner is to discover, first, the hardness or resistance to pressure; second, the consistency and form of the organs

which he can touch; and, thirdly, whether any swellings which he feels are movable, bound down and immovable, pulsating, soft or hard, nodular or smooth. The patient whose abdomen is to be palpated must be placed flat on his back with the knees drawn up to relax the abdominal muscles, the head and neck should be raised, and, if possible, the attention of the patient should be diverted by conversation about some symptom which exists elsewhere than in the belly while the examination is made, as in this way voluntary muscular resistance is removed to some extent. He should be made to breathe easily through his opened mouth; and if the belly-wall remains so rigid that a perfect examination is impossible, and yet the results of such an examination are very important, ether or chloroform should be given to relax the muscles.

Great resistance of the rigid abdominal muscles is found whenever peritonitis is present in an acute form, in some cases of renal and hepatic colic, and more commonly in lead colic and in hysteria. In peritonitis great tenderness to the slightest touch is also present. Another symptom of acute peritonitis, aside from the exquisite tenderness of the abdomen, the drawn lip, the thirst, and the distention or rigidity of the belly-wall, is pain of a severe character; unless it be septic peritonitis, when pain may be absent. There are also the drawing up of the limbs to relieve abdominal tension; obstinate constipation, moderate fever, and a very rapid, quick pulse. The tongue speedily becomes dry and parched, and collapse may speedily ensue in severe cases. It is not to be forgotten that localized peritonitis may result from many causes, usually from disease of the appendix vermiformis or the genito-urinary tract in women, and that the local symptoms and lesions may be limited by a wall of lymph to a very small area of the abdominal cavity.

It must be remembered, however, that the anterior abdominal wall, particularly that of nervous persons, is often very sensitive or "ticklish," and the mere exposure of the skin to the air of the room, coupled with the fear of the examination, may cause great rigidity of the belly-wall without there being any abnormal condition present. This can generally be overcome by gentleness in palpation and by resting the palm of the hand on the belly and partly flexing the fingers, rather than by attempting to insert the finger-tips between the abdominal muscles. The writer has recently seen a case of rhythmical hysterical spasm of the recti muscles in a

male, which at first gave the sensation of an enormous diffuse pulsating aneurism of the abdominal aorta.

Let us suppose that on placing the hand upon the epigastrium and the upper part of the umbilical area that we find a swelling. In the first place, we must decide as to whether it is in the abdominal wall or in the abdominal cavity. If it is in the wall, it will be movable with the tissues of the wall and readily grasped by deep palpation; but if in the abdominal cavity, the abdominal wall may be made to move over it, unless it be attached to the parietal peritoneum. Let us suppose it is in the wall of the abdomen, what can the swelling be? It may be a fatty tumor; in which case its surface will be dimpled and resistant, probably not painful, unless the part has been inflamed by rubbing or an injury, and it will not fluctuate. There will generally be a history that the person has exercised constant pressure on the part, as in leaning against a bench or table. Again, it may be an abscess; but aside from the rarity of this condition, we can exclude such a possibility by the absence of pain, fluctuation, and the history of a severe injury.

Very much more commonly a swelling in the epigastrium, or upper umbilical area, is due to an intra-abdominal cause. In adults the most common cause is probably a growth (generally a carcinoma) of the pyloric end of the stomach. In other instances it is due, particularly in children, to enlarged lymphatic glands, as in tubercular disease of the mesentery. This is also sometimes seen in adults. Sometimes by reason of tubercular peritonitis a nodular mass is not only felt in this area, but an abscess containing tubercular pus may be formed and become surrounded by walls formed by the gluing together of the organs by lymph. Carcinoma of the pancreas may also cause a swelling in this neighborhood, or a cyst of the pancreas may be present. Aneurism of the abdominal aorta is also not to be forgotten. Sometimes, too, a distended or carcinomatous gall-bladder may project into this area.

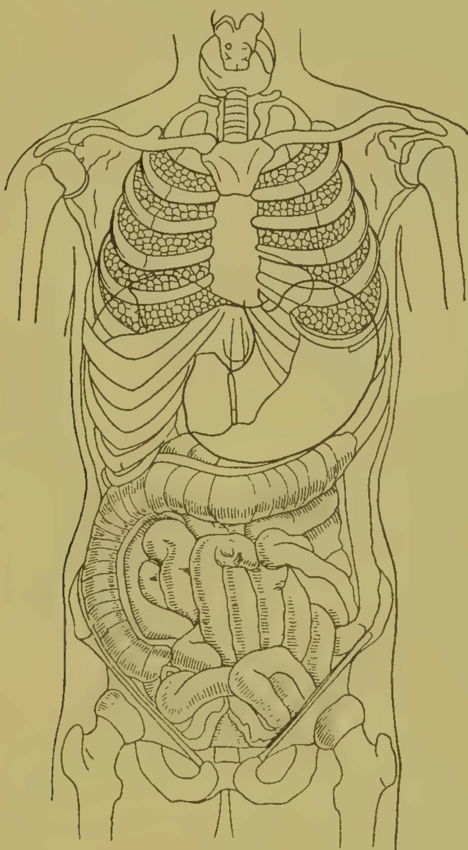
If the growth be gastric carcinoma, the patient will be in or past middle life (probably between the fortieth and seventieth year); will have a history of constantly increasing pain and discomfort in the stomach; there will have been much sour belching, and perhaps vomiting of coffee-ground-looking material; marked loss of flesh and some cachexia will be present. According to Welch's statistics, out of 1300 cases of gastric cancer, 791 were in the pylorus, 148 in the lesser curvature, 104 in the cardia, 68 in the posterior wall,

and 61 involved the whole stomach. The remainder were in the fundus, the greater curvature, or the anterior wall. The growth, if in the pylorus, is usually freely movable, and for this reason can be readily felt, and then is often momentarily lost to palpation. Its position is apt to change with the posture of the patient and the presence or absence of food in the stomach. Pain is usually elicited on deep pressure, and, if the growth be large and at the pylorus, the symptoms of dilatation of the stomach will be present, because that viscus is dilated through the obstruction of the pyloric opening, which results in retention of the gastric contents. Under these circumstances, whatever the cause of the obstruction may be, or if the gastric dilatation simply results from inherent feebleness of the stomach-walls, the entire upper part of the abdomen will be found distended, tense, but yielding, and the history will show that the patient is attacked now and again by vomiting, during which the most extraordinary quantity of food and liquid, which has gradually accumulated, will be expelled. The probable diagnosis of gastric cancer and of gastric dilatation can be usually confirmed by percussion, after distention of the stomach, and by the use of the gastrodiaphane. (See early part of this chapter.)

Even before the stomach is artificially distended with gas percussion will give us valuable information in this connection, for, if obstruction of the pylorus exists, there will be found either a large area of gastric tympany through the accumulation of gas from fermentation, or, if no vomiting has taken place for some time, an equally great area of gastric dulness due to an accumulation of food and liquid. If in such a case we first wash out the stomach by means of a stomach-tube and then fill it with gas by giving the patient to drink, first, a half-glass of water with sodium bicarbonate in it, and then another half-glass with tartaric acid dissolved in it, so that gas will distend the viscus, we shall be able by means of percussion to outline the stomach with ease. It is best to mark the edge of gastric resonance by means of a blue pencil and thus to map out the gastric area. If there is a growth at the pylorus, causing obstruction, there will be impairment of resonance wherever the pylorus may be situated. While this is a somewhat indefinite statement, it is to be remembered that a more definite one is liable to mislead the student, for even in health the position of the pylorus changes greatly when the stomach is empty or filled with food. Thus when empty the viscus hangs with the pylorus very low, but

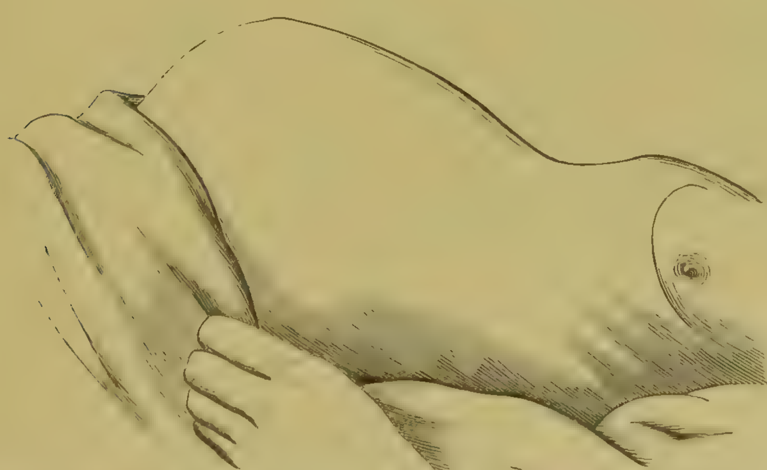
when it is filled the pylorus is raised. Fig. 136 shows the normal gastric area when the stomach is distended with gas. Fig. 137, taken from Osler's *Lectures on Abdominal Tumors,* illustrates the

FIG. 136.



Outline of normal position and size of stomach in an adult when distended with gas. (After MEINERT.)

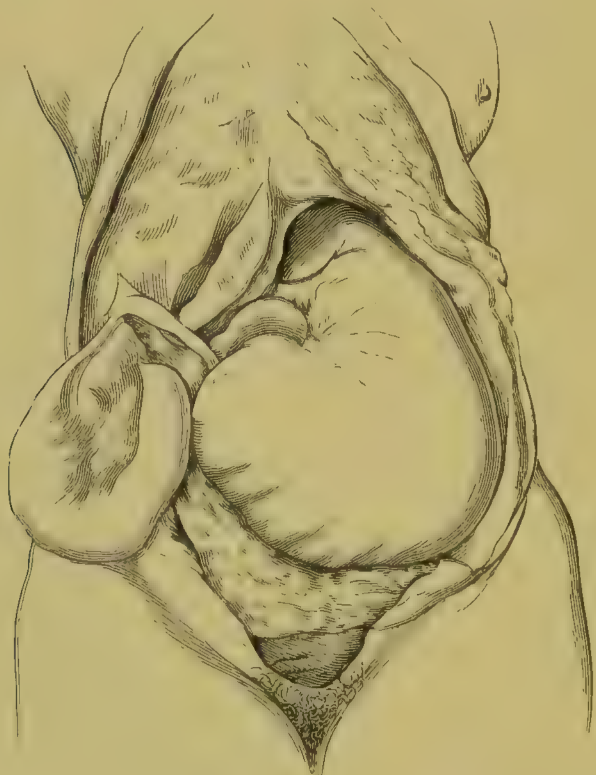
FIG. 137.



Profile view of the abdomen of a woman aged sixty-five years, showing the tumor formed by the dilated stomach. (OSLER.)

extraordinary descent of the stomach made in some cases of gastric dilatation in the adult. (Figs. 137, 138, and 139.)

FIG. 138.



Showing the position and size of the stomach. (OSLER.)

FIG. 139.

Tumor of the abdomen caused by a dilated stomach. (OSLER.)

Many of these cases of gastric dilatation are also associated with atrophy of the gastric tubules, or at least an absence of any secretion of normal gastric juice. The matters vomited, or washed out of the stomach, are often devoid of hydrochloric acid, but loaded with an excess of lactic acid. Lactic acid is tested for as follows, the hydrochloric-acid test being given below: A few drops of neutral ferric chloride solution are mixed with one or two drops of pure carbolic acid, or 10 c.c. of a 5 per cent. solution of carbolic acid, and water added until an amethyst hue develops. A few drops of the filtrate derived from the stomach-contents are now added, and if lactic acid or lactates are present the amethyst-blue will become yellow in color. This is a very delicate test for lactic acid.

Sometimes in cases of chronic gastric ulcer the area involved becomes so indurated as to be felt as a hard mass in the stomach-wall. In such instances the points which aid us in separating the condition from gastric cancer are the facts that the patient is young and usually a woman; that the vomiting occurs immediately after taking food, for in gastric cancer it is only seen in most cases several hours after food has been taken; that there is no sign of gastric obstruction; that there is an excess of hydrochloric acid in the gastric contents in cases of ulcer, and an absence of HCl in cases of cancer; and, finally, that there is no cachexia in cases of gastric ulcer, though there may be anæmia. There is usually in cases of ulcer no great loss of weight, unless the symptoms have been present a long time.

In cases of gastric ulcer great pain is often produced by deep or even superficial pressure over the epigastrium and a painful spot can generally be found on the back, about the angle of the right scapula.

There is no better place than the present to speak of the manner of testing the stomach-contents for hydrochloric acid. The patient is directed to take no food for at least twelve hours before presenting himself to the physician. On his arrival for examination he is given what is known as "Ewald's test-breakfast," which consists of an ordinary dry roll and a little over half a pint of water which has been warmed, and he is directed, after swallowing these materials, to wait for an hour. The stomach is now emptied by the introduction of the bulbed stomach-tube, and the gastric contents filtered. A few drops of a solution of phloroglucin and vanillin are next placed in a porcelain dish and a few drops of the gastric liquid are allowed to trickle down to the edge of the solution. The porcelain dish is

gently heated over a spirit-lamp or Bunsen burner, and if hydrochloric acid is present there will appear a red tinge, which is an absolute proof of the presence of hydrochloric acid.

The solution of phloroglucin and vanillin is made as follows:

Phloroglucin	gr. xxx.
Vanillin	gr. xv.
Absolute alcohol	f℥j.

This solution is pale yellow in hue. It must be kept in dark bottles, as on exposure to the air and light it becomes brown and worthless.

If the cause of the swelling of the abdominal wall be tubercular glands, they will be found, in all probability, on deeper and more general palpation, to be scattered all through the abdominal cavity; there will be a history of alternate constipation and diarrhœa, of fever, of general loss of strength, of loss of appetite, and examination of other parts of the body may reveal the signs of tuberculosis elsewhere.

The presence of a resisting mass, deeply situated in the epigastrium, or the upper part of the umbilical area, and felt only on deep palpation, and then often indistinctly, should bring before the mind the possibility of the presence of carcinoma of the pancreas, a diagnosis which will be largely confirmed if cachexia be asserting itself, if there be great pain in this neighborhood, and if there are oily stools after fats are taken, as a result of the absence of pancreatic juice. Still further confirmation of this diagnosis will be present if diabetes mellitus be developed by the patient (pancreatic diabetes). Such a growth in the pancreas is usually scirrhus cancer and may be primary or secondary. Segré found that of 627 cases of carcinoma of the upper abdominal organs cancer of the pancreas occurred in 127, but only in 12 of these primarily. Stiller asserts that the following symptoms are fairly sure signs of pancreatic cancer, namely, marked dyspepsia, rapid emaciation and cachexia, subnormal temperature, persistent and progressive jaundice without hepatic enlargement, but often with swelling of the gall-bladder from obstruction to its duct. These signs are, of course, only of value if the evidence of malignant growth elsewhere can be excluded. Very rarely swelling of this region, either rapid or slow in onset, follows upon the formation of cysts in the pancreas, as a result of obstruction of the duct of the gland. When they occur these cysts may quite fill the abdominal cavity, although, as a rule, they are quite small. As

pointed out, however, by Jordan, the real cause of swelling in the pancreatic region may be hemorrhage into the lesser peritoneal cavity. He summarizes some of his views in regard to this matter as follows:

"Contusions of the upper part of the abdomen may be followed by the development of a tumor in the epigastric, umbilical, and left hypochondriac regions. Such tumors may be due to fluid accumulations in the lesser peritoneal cavity, and when the contents are found (on aspiration) to have the power of converting starch into sugar we may assume that the pancreas has been injured." Finally, Jordan states that "many such tumors have been regarded as true retention-cysts of the pancreas."

In other instances a swelling in this neighborhood may be due to what is called *pyo-pneumothorax subphrenicus*, a condition of abscess in the peritoneal cavity below the diaphragm, produced by perforation of the stomach or transverse colon. The abscess so produced may contain gas, and for this reason the swelling may be quite resonant on percussion. Abscess in this region also follows abscess of the pancreas, or fat-necrosis of this organ in rare instances.

Sometimes, too, we have marked enlargement of the head of the pancreas, due to a malignant pancreatitis. This is particularly apt to be associated with cholelithiasis.

The appearance of sudden swelling in the neighborhood of the pancreas, associated with intense pain, nausea, and vomiting, may be due either to acute hemorrhagic pancreatitis, to intestinal obstruction, or to acute peritonitis, resulting from perforation. The last two are the more common. An exploratory operation is the only way of deciding the diagnosis positively. (See chapter on Vomiting for symptoms of intestinal obstruction.)

Aortic pulsation is often transmitted to the hand by enlarged abdominal glands or tumor-masses. If the pulsation of the aorta is not transmitted by glands or tumors, it may be due to aneurism of the abdominal aorta, the diagnosis of which is established, if, in addition to a pulsating sensation, we also find on palpation a marked thrill, an expansile movement of the tumor, and on auscultation we hear a bruit. Pain due to the pressure of the aneurismal sac upon some of the nerves of the abdominal cavity may also be a prominent symptom.

Localized swellings due to other causes than those already discussed are due to impaction of feces, volvulus, and intestinal obstruc-

tion from other causes, as, for example, cancer of the bowel, which occurs most frequently in the cæcum, when the growth will be found in the right groin, or in the sigmoid flexure, when it will be found in the left groin.

Tumors or foreign bodies in the bowel can nearly always be moved about, unless bound down by inflammatory adhesions, so differing from growths which involve the immovable parts, such as the retroperitoneal glands. Very rarely we find cancerous tumor of the omentum, which usually becomes retracted and indurated so that its hardened edges can be felt extending across the abdominal cavity. More commonly multiple nodules in the omentum, or studded over the surface of the bowels, are due to peritoneal tuberculosis. Not rarely these nodular masses are studded over the mesentery.

Floating kidney may also cause a marked movable swelling or tumor-like mass in the upper zone of the abdomen. If the belly-walls are thin, the kidney-shape can sometimes be outlined by palpation and even the pulsation of the renal artery can be felt; but, as a rule, this cannot be done, and the dilatation of the pelvis of the kidney by the obstruction of the ureter, which has become twisted, may distort the shape of the organ. Deep palpation of the flank, if the kidney has floated away from its normal seat, may reveal lessened resistance in this area, and bimanual palpation, one hand being placed at the back and the other in front, may reveal the shape of the organ elsewhere. Further, if the patient be made to lie on the side the dislocated kidney may sometimes be clearly outlined by bimanual palpation.

When the kidney is enlarged from cystic degeneration, from ordinary hydronephrosis, and from echinococcus cysts, it may be readily felt in the umbilical area in many instances. Hydronephrosis has been mistaken, in children particularly, for sarcoma of the kidney, and in adult females for ovarian tumor. The diagnosis can only be made in some of these cases by tapping. The fluid obtained in hydronephrosis will usually be somewhat turbid and contain epithelial cells. It should not be forgotten that the condition of hydronephrosis may be intermittent, for, if this is not remembered, the physician may be misled into thinking that the disappearance of the swelling is due to a floating kidney slipping back into its place.

Bulging of the flank, with pain, fever, and perhaps fluctuation,

indicates perinephritic abscess or caries of the spine with cold abscess.

Peristaltic movements of the intestines can sometimes be felt through the belly-walls, and, if the contraction of the muscular fibres is excessive, nodular masses of momentary existence may be caused. Closely associated with this sensation on palpation is what is called "phantom tumor." Such a formation is generally found in hysterical women and often leads to ludicrous errors in diagnosis. It is due to persistent dilatation of a knuckle of intestine by gas, thereby forming a moderately hard, and more or less constant, mass which may resemble a real tumor. Examination of the patient under ether will usually reveal its true character. Localized superficial and inconstant tumors may arise through spasmodic but localized contractions of the recti muscles.

Finally, a swelling in the neighborhood of the umbilicus should always arouse the suspicion of an umbilical hernia, and the situation of the swelling at the umbilicus, the fact that percussion over it gives a highly tympanitic note, owing to the gas in the prolapsed gut, and the possibility of reducing its size by taxis, will render a diagnosis of umbilical hernia possible.

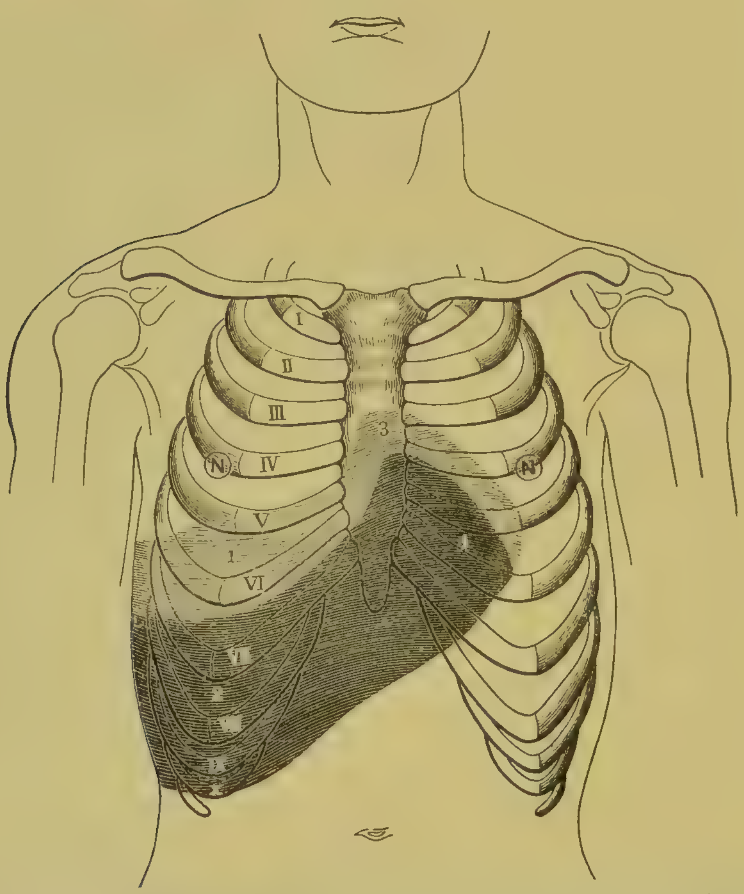
If on palpating the epigastrium and umbilical area nothing abnormal has been found, we next seek to discover if there is anything abnormal in the right hypochondrium, or, in other words, whether there is any disease of the liver.

Normally, in the adult, this gland cannot be felt below the ribs, except part of the left lobe in the epigastrium occasionally. Sometimes, on deep inspiration, the diaphragm pushes the liver low enough for it to be felt. In children the liver is naturally large enough to be felt below the ribs.

When the normal liver is percussed we find that it lies in the area shown in Fig. 140, and that as we percuss above it on the ribs in the mammary line we first get pulmonary resonance, then a little below this impaired resonance, due to the fact that the lower edge of the lung is interposed between the chest-wall and the liver; and still lower we find absolute dulness or flatness due to the solid liver itself. Below this area, which ceases just below the lowest rib, we usually find tympany on percussion, due to the gas-distended bowel. If we percuss in the midsternal line, we get the same signs, but they begin as high as the nipple, or above it, and then cease on a line drawn across the abdomen about midway between the ensiform

cartilage and umbilicus. To the left of the middle line of the sternum the liver-dulness merges into the cardiac dulness. (See Fig. 140.) In the mammary line liver-dulness begins at the fifth rib, laterally it begins at the seventh and eighth, posteriorly at the tenth, owing to the sloping position of the diaphragm.

FIG. 140.



Showing absolute and relative percussion-dulness of liver and heart. 1. Relative dulness of liver. 2. Absolute dulness. 3. Relative dulness of heart. 4. Absolute dulness.

When a hard and firm mass, with a smooth surface, can be felt in the right hypochondrium or right umbilical area, which is movable, and which has an edge which can be readily felt on deep palpation, the mass is probably an enlarged liver or one pushed down into the abdominal cavity by a large pleural effusion or a subphrenic abscess, or sometimes by an emphysematous lung. The causes of enlargement are amyloid degeneration, congestion, hypertrophic cirrhosis and abscess, carcinoma, sarcoma, and lymphadenoma. When the surface is found to be smooth the condition is probably amyloid

or fatty degeneration, or congestion. If the surface is rough, it will probably be due to cirrhosis, which gives a granular sensation to the hand when the abdominal wall is moved over the organ. In malignant growth large and small nodules may often be found, and depressions or umbilications of its surface may be marked. When, on palpating the liver, we find marked tenderness and some swelling, and, associated with these symptoms, fever, rigors, sweats, and sometimes vomiting, and, in addition, a history that the patient has had dysentery or has had exposure to tropical heat or has swallowed much bad water, we are forced to the belief that an abscess of the liver exists. This may be single or multiple. If the latter, it is probably due to pyæmia, and no spot of fluctuation will be found as a rule; whereas, if it is large and single, fluctuation is sometimes felt. Further, the enlargement of the liver in the pyæmic form is uniform, whereas in the single abscess there is often one spot which is swollen or enlarged. If a single large pyriform swelling, which is yielding and somewhat painful on palpation, be found, and there is some fluctuation present, abscess must be thought of, or in its place impaction of the gall-bladder with gall-stones or its distention by obstruction to its duct. The history of the case will usually separate the conditions, one from the other, for diagnostic purposes, for in the case of abscess the history will probably be that of a person exposed to tropical heat or one who has had an injury, an acute infection, or an amœbic dysentery, while if gall-stones be the cause of the swelling there will be a history of gall-stone colic, of jaundice, or of hepatic fever. More rarely a single hepatic swelling may be due to hydatid cyst, but the history and presence of fluctuation, combined with the result of examining the fluid aspirated from the swelling, will decide the diagnosis.

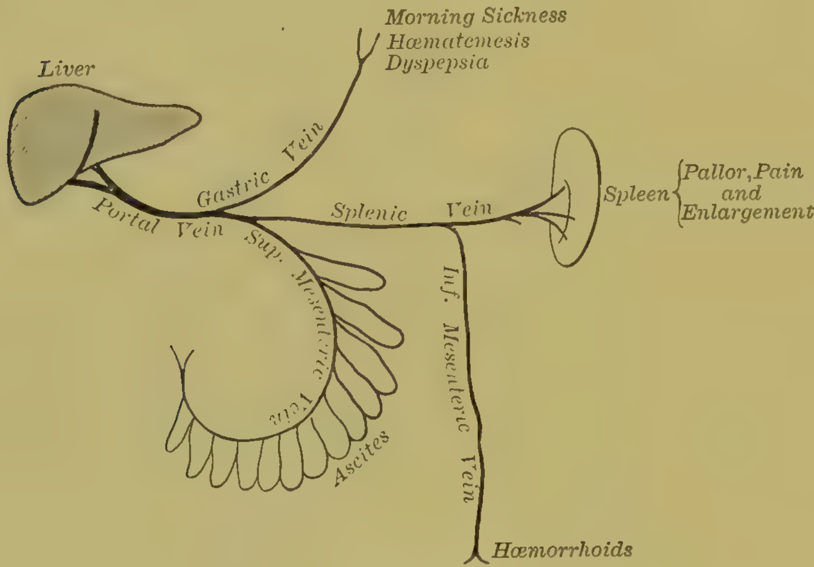
The consistency of the liver is usually very hard in cases of cirrhosis, carcinoma, and amyloid degeneration. In cirrhosis there will be some ascites in many cases, some swelling of the legs perhaps, and dull pain in the hepatic region. The digestion will be disordered, there will be marked loss of flesh and often hæmatemesis. Sometimes coma comes on. In the malignant disease there will be pain, marked emaciation, and cachexia; nodules will be felt in the liver-substance, and the organ be found much enlarged. Tenderness on pressure will be marked. Sometimes ascites will be present and a growth may be found, usually as the primary lesion, in the stomach or bowel. In the case of amyloid liver there will be a history of prolonged sup-

puration elsewhere, and there will be present disordered digestion, irregular bowel-movements, and little pain.

Marked tenderness of the hypochondrium is usually found in congestion of the liver, in inflammation of its tissues, such as that caused by an infection or by gall-stones in its substance, and in malignant growth. Tenderness is practically absent in waxy liver and in fatty degeneration.

In cases of cirrhosis of the liver, whether it be in the hypertrophic or atrophic form, the organ presents no symptoms in itself save that in the hypertrophic state its size is increased so that it can be felt below the ribs, whereas in the atrophic state it cannot be felt except

Fig. 141.



To illustrate symptoms of cirrhosis of liver.

by pushing the fingers well up under the ribs. The symptoms accompanying cirrhosis are chiefly connected with disorders of the alimentary canal, either through direct failure in the digestion and assimilation of food, or from changes in the blood-supply of the abdominal contents. The following excellent diagram from Taylor's *Index of Medicine* shows what these symptoms are, and discerns their cause at the glance, the cirrhotic process, of course, obstructing the flow of blood in the liver. (Fig. 141.)

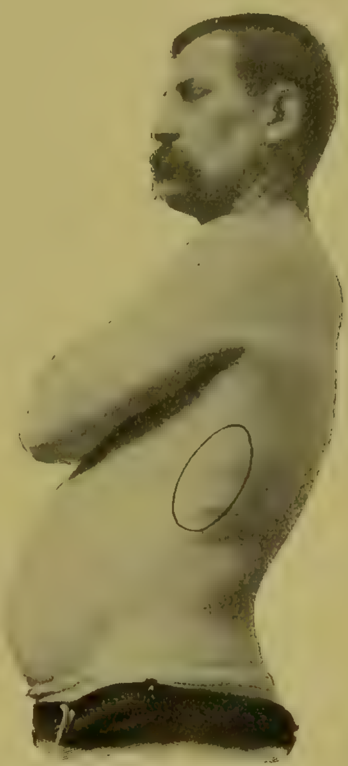
Finally, the physician who finds the lower margin of the liver abnormally low down in the abdominal cavity should not make a diagnosis of enlargement of this organ until he has assured himself that the extension of the margin of the liver is not due to an effusion

in the right pleural cavity which presses upon this organ. So, too, if the patient is a woman, the lower border of the liver may have been pushed down by tight lacing, and careful palpation may reveal a furrow across its surface produced by the corset.

A small pear-shaped mass protruding from under the liver is usually due to an enlarged gall-bladder, distended by bile or calculi. If the former, pressure may cause it to disappear owing to the bile being pressed out into the intestine.

In the left hypochondrium the spleen can be very readily outlined by percussion, in persons not inordinately fat. Its normal position is best shown in the accompanying figure, 142.

Fig. 142.



Normal position of the spleen.

The upper border of the spleen is on a level with the tenth dorsal vertebræ and the lower border on a level with end of the eleventh rib. Its upper edge or limit is on a level with the ninth rib. In percussing the spleen heavy percussion is to be avoided, since this may develop the resonance of the stomach or bowels. The spleen cannot be palpated unless greatly enlarged, but it may be found bulging from beneath the lowest rib in typhoid fever; in scarlet fever; as the result of acute or chronic malarial fever; in leucocythæmia of the spleno-medullary variety; in amyloid disease, as that after long suppuration; in early syphilitic infection; and in any disease which causes venous engorgement of the abdominal viscera, such as cardiac disease or hepatic cirrhosis. Sometimes displacement of the spleen downward arises from emphysema of the lungs or left-sided pleural effusion.

Acute splenic swelling sometimes comes on in cases of general septicæmia.

Nearly always the splenic surface is smooth, except for the notch in its surface (see Fig. 133, p. 306), unless the disease be the rare condition of hydatid disease or carcinoma.

There yet remains for discussion the significance of increased

resistance on palpation, and percussion-dulness, in the groins. In the right iliac region the presence of swelling, increased resistance, impaired resonance, or tympanites, particularly if pain and tenderness are present, point strongly to appendicitis or to inflammation about the *caput coli.* Sometimes, however, the presence of a distinct lump in this region in a person advanced in life may mean a malignant growth, for carcinoma of the caput coli is not rare.

If the left groin is affected in a person well advanced in years, carcinoma is also to be regarded as possible, for the sigmoid flexure is a frequent seat of such growths. In a young person, or a child, impaction of feces, a foreign body, or intestinal obstruction is to be considered. (See chapters on Vomiting and on the Bowels.)

For the diagnosis of renal diseases reference is to be made to the chapters on the Bladder and Urine, on the Blood, on the Bloodvessels and the Pulse, and that upon the Thorax, that part on the heart; the chapters on Vomiting and on Headache, and to those on Coma and Unconsciousness, and Convulsions and Spasms.

For further information in regard to the diagnosis of diseases of the abdominal viscera, the reader is referred to the chapters on the Skin, that part on jaundice; the chapter on Vomiting, part on intestinal obstuction; that in the Bladder and Urine; and that upon the Bowels and Feces.

CHAPTER X.

THE BLOODVESSELS AND PULSE.

The condition of the bloodvessels on palpation—Feeling and counting the pulse—The quality, force, and volume of the pulse in health and disease

ONE of the first things that the physician does when he is studying the condition of a patient is to feel the pulse, even if the symptoms which are present do not indicate circulatory disturbance, because the pulse is an index of the condition of the heart as to its power, its valvular action, and its nervous state. The pulse very often gives us information of the presence of renal disease, and it will frequently give us a general idea of the tone or degree of debility of the patient. By feeling the pulse we also gather valuable information as to the condition of the arteries, and this is a very important part of the diagnosis, for, to use an old saying, "a man is only as old as his arteries," and if he is sixty years of age and has good vessels he is, as a rule, as young in health as another man of thirty with bad vessels, because it is by the bloodvessels that the tissues of the body are nourished, and, as life depends upon this process of nutrition, the better the vessels the better the vitality.

When examining the pulse of a patient who is well enough to be up and about the physician should wait until sufficient time has elapsed after exercise for the pulse to become steady, and the patient should be in a sitting or reclining posture in order to prevent overaction of the heart. Particularly is it important in the case of nervous individuals to wait for sedation to follow the excitement of meeting the physician.

Often when called to see a sick child or a nervous woman, who may be sleeping at the moment of the physician's arrival, a true estimate of the pulse can be made without disturbing the patient by gently putting the tip of the finger on the temporal artery where it passes over the zygomatic process. This artery may also be used for this purpose in cases of tremor, chorea, delirium, or mania, where the hand is constantly moved about so that the radial artery cannot be felt.

In counting the pulse it is best to count it for the entire minute, or to count it for fifteen seconds and then multiply the result by four. If the pulse is irregular, it is always best to count it for a minute. If the pulse be very irregular and running, and so difficult of counting, the count should be made by listening at the præcordium for the apex-beat.

Before considering the qualities of the pulse in health or disease, it is well to understand what it is due to and the manner in which the circulation is carried on. The bloodvessels consist of the arteries, arterioles, capillaries, venules, and veins. These vessels always contain blood during life, and the function of the heart is to propel the blood through them. The flow of blood is maintained, first, by the force expended by the heart, and, second, by the tonicity of the bloodvessels. If the bloodvessels of the body become relaxed, as in death, all the blood is readily held by the ones most relaxed, namely, the abdominal, thoracic, and other veins. We find therefore that the vessels are only filled with blood when their walls are to a certain extent constricted by the contraction of their muscular fibres ; and that this contraction is maintained by the action of the vasomotor centre in the medulla oblongata, which also controls many minor centres governing small areas of vessels. The arteries are very elastic in health, and when filled with blood are slightly distended. Behind the column of blood, which being a fluid confined laterally is practically a solid, for fluids are incompressible, is the heart, and in the arterioles are muscular fibres, which by their contraction regulate the flow of blood into the capillaries, from which the nutritional processes are carried on. The blood in the arteries is, therefore, subjected to three chief pressures, namely, that of the heart behind the column, that of the elastic and distended arterial walls on the sides of the column, and the resistance of the contracted arterioles in front of the column. By these means blood-pressure or tension is maintained. If the heart beats more strongly or the arterioles contract more tightly than normal, the blood-stream is under a greater pressure than before. If the heart is feeble or the arterioles lax, the pressure falls, because the blood is not pressed upon behind or obstructed in its flow in front. If the tension is above or below normal, the interchange of food and oxygen and carbonic acid between the tissues and the blood in the capillaries is perverted, for the rate of flow in the capillaries depends largely upon the blood-pressure in the arteries. Now the capacity of the capillary system of vessels is many

times greater than that of the arteries, and, if the arterioles relax, the capillaries and veins will retain all the blood, and in them it will stagnate and become useless.

The manner in which arterial tension is chiefly maintained having been described, we can now consider the pulse-beat itself. The individual pulse-beat is not a wave of blood sent out by the heart, but it is the transmission of the force of the heart-beat sent along the blood-column, and the character of the beat gives us, therefore, an idea of how forcibly the heart is driving another quantity of blood into the aorta, and also how much blood is being sent out at each beat.

Supposing, therefore, that on feeling the radial pulse we find that the artery is tense and hard, and that the individual beat is strong and its volume great, this signifies that there is an excited vasomotor centre, causing contraction of the vessels, and that an excited, over-acting heart is forcing the blood into the already tense vessels. If this condition increases, one of three things can happen, either the heart will be unable to pump the blood out into the arteries against the pressure and consequently become distended and paralyzed, or the bloodvessels will burst in the weakest spot, or the spasm of the arterioles will have to give way. It is the first result which we meet in cases of true angina pectoris, for in this state we find great arterial tension, with distention and engorgement of the left side of the heart, and the moment nitroglycerin or nitrite of amyl relaxes the spasm of the arterioles the symptoms are relieved. It is the second result which often produces apoplexy by rupture of the weakest vessel, usually the middle cerebral artery. It is the third result which we try to bring about for the relief of the patient, either by drugs or by bleeding. Where we have atheroma or hardening of the bloodvessels as the result of old age, syphilis, or chronic vessel-changes the very inelasticity of the vessel, associated, perhaps, in some cases, with some irritability of the vasomotor centre, causes a high arterial tension, with a laboring heart, a congested head, and a feeling of fulness of the head, of which the patient will seriously complain. The second sound of the heart will also be much accentuated.

The discovery of a high arterial tension in a young person, or in an older one who has not atheromatous vessels, will generally mean the presence of an excited circulation, in connection with some acute inflammatory disease in its early stages, and, if high fever is present

in a previously healthy person, the pulse will be found to be quick and hard.

The condition of intense vascular relaxation, due to failure of the heart or the arterioles to maintain blood-pressure, is seen in cases of fainting and syncope on the one hand, or of collapse and shock on the other. Here we find a soft, easily extinguished blood-stream, which can, by pressure on the artery, be readily cut off from the distal vessels. The artery feels relaxed to the physician's finger and the skin may be bedewed with sweat.

We can conclude, therefore, that high arterial tension indicates in the young, as a rule, an excited circulation, due to some acute ailment in its early stages, or, if in an older person not suffering from an acute malady, it is due to atheroma of the bloodvessels, renal disease of a chronic interstitial type, or hypertrophy of the heart, provided, of course, in all cases that there is no history of the recent ingestion of powerful stimulants to the circulation.

A very low arterial tension indicates a feeble condition of the system, such as is seen in all exhausting diseases, acute or chronic, or, if no disease be present, in the sense of an acute malady, it indicates general nervous debility, with or without the presence of a feeble and dilated heart.

The pulse itself varies as to volume, character, rapidity, and force, and does so within normal limits, and still more so under the effects of disease. It varies greatly according to age. Thus, the pulse of the newborn child is usually about 135 to 140,, at one year 120 to 130, at two years 105, at four years 97, at ten years about 90, at fifteen years 78, and from twenty to fifty years at 70 per minute. At eighty years of age it is usually about 80 beats per minute. The rate is also increased by taking food, by exercise, nervousness, and by pain and fever, as will be stated again in a moment.

The volume of the pulse-wave depends chiefly upon the quantity of blood expelled from the heart at each systole, and also upon the condition of the aortic valves of the heart, in so far as their ability to prevent regurgitation is concerned. Stimulation of the vagus nerves usually results in a large pulse-wave, as already pointed out, as does also cardiac hypertrophy with dilatation. If, on the other hand, part of the blood thrown out of the heart into the aorta falls back into the ventricle, we have a pulse of small actual volume, and this is called, because of the peculiar sensation which it gives to the finger, the "trip-hammer," "water-hammer," or "Corrigan's pulse." In

such a case because of the power of the ventricle the blood is forced out into the aorta under great pressure, but as the last part of the wave regurgitates the pulse is found to be short and sharp. In mitral regurgitation or in mitral stenosis the pulse is usually small in volume, because the left ventricle has not, or cannot get, enough blood at each beat to send out a voluminous wave.

So far as the character of the pulse is concerned we recognize one which is slow and full, as that seen after digitalis is used; that which is short and sharp, as in aortic regurgitation; that which is small and hard, as is often seen in aortic obstruction; and the small, wiry pulse of acute peritonitis.

FIG. 143.

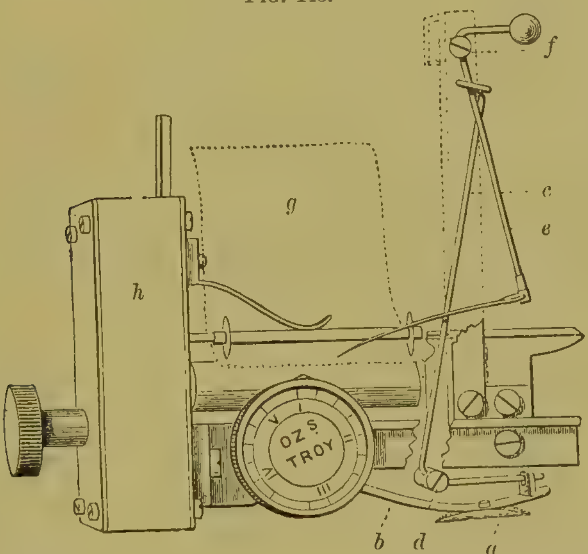


Diagram of a sphygmograph. (DUDGEON'S.) Certain supporting parts are omitted so that the multiplying levers may be displayed. *a* is a small metal plate which is kept pressed on the artery by the spring *b*. The vertical movements of *a* cause to-and-fro movements of the lever *c* about the fixed point *d*. These are communicated to and magnified by the lever *e*, which moves round the fixed point *f*. The free end of this lever carries a light steel marker which rests on a strip of smoked paper, *g*. The paper is placed beneath two small wheels and rests on a roller which can be rotated by means of clock-work contained in the box *h*. The paper is thus caused to travel at a uniform rate. The screw graduated in ounces (Troy) is brought to bear on the spring *b* by means of a cam, and by this the pressure put on the artery can be regulated. The levers magnify the pulse-movements fifty times.

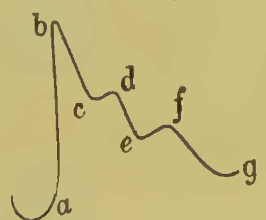
Various names are applied to a pulse possessing certain peculiarities. Thus, we have under the name *pulsus paradoxus* a pulse which disappears with each deep inspiration. It is usually due to indurative mediastino-pericarditis, whereby inflammatory bands press on the bloodvessels or the heart. If the beats of the heart are irregular in force, but regular in rhythm, we have developed a *pulsus alternans*.

A dicrotic pulse is one which is characterized by a reduplication, which feels like a second beat following the first before the latter is over. It is found in many cases of exhausting fever, and depends upon an undue elasticity of the bloodvessels, with relaxation of the arterioles, so that the blood first unduly distends the arteries, which then contract upon it, and thus produce the second wave or apex to the pulse-curve.

We can study the pulse either by the touch or by the sphygmograph. If by the latter means, the instrument of Dudgeon is the best. (Fig. 143.)

The normal pulse-wave is shown in Fig. 144.

FIG. 144.



ab. Percussion up-stroke. *abc.* Percussion-wave. *cde.* Tidal wave. *efg.* Dicrotic wave. *def.* Aortic notch. *fg* Diastolic period.

It will be seen that there is a distinct upstroke produced, which is called the line of ascent. This is due to the distention of the artery produced by the ventricle forcing blood out into the aorta. There is after this a line of descent interrupted by two separate secondary waves, which are called catacrotic waves. The second or lower of these is called the dicrotic wave, and is the one which becomes marked enough to be felt in some cases of disease. The duration of the period of descent corresponds to the time the blood is flowing out of the arteries into the capillaries, and, if this flow is rendered difficult by vascular spasm, the line of descent will be gradual. If easy from vascular relaxation, it will be short. If the drop is very sudden, it is pulse of "empty arteries," so-called, as after severe hemorrhage in cases of acute regurgitation.

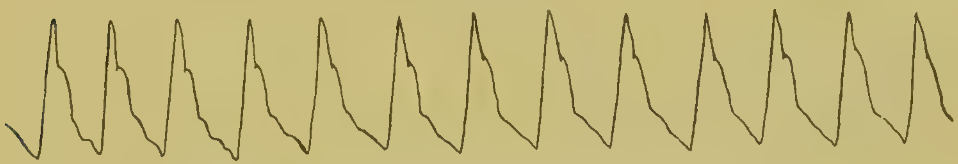
Very small irregularities of the line of descent are due to the elastic bloodvessel being thrown into vibrations by a forcible pulse-wave.

In Fig. 145 is shown the typical pulse-wave of aortic regurgitation; and in Fig. 146 that of mitral stenosis, which is irregular in time and volume.

The rapidity and force also depend largely on the condition of

the bloodvessel-walls, particularly the rapidity. The latter also depends upon the activity of the pneumogastric nerves in regulating the beat of the heart. Thus, if the arterial pressure be very high, through spasm of the arterioles, the difficulty experienced by the heart in forcing blood into the arteries will be so great that pulsa-

FIG. 145.



Tracing from a case of aortic regurgitation. (MUSSER.)

FIG. 146.

Tracing from a case of mitral stenosis, showing increased tension and some irregularity. (MUSSER.)

tion will be very slow; whereas, if the normal resistance to the action of the heart be removed by vasomotor relaxation, the beat will be rapid, just as the wheels of a locomotive fly around on a slippery track when the friction or the resistance is removed. If the vessels are relaxed, the impetus communicated to the column of blood in the vessels by the heart is lost, and so the pulse is not forcible; or if the resistance is excessive, the force is dissipated.

The vagus or pneumogastric nerves are continually holding the heart in check, and by causing full diastole enable it to send out a large wave of blood at each contraction. If they are greatly stimulated, we have a very slow pulse and a full wave of blood with each heart-beat; but as the heart now beats very slowly the blood-pressure may fall for lack of blood in the vessels, unless there is an increased force of the heart at each contraction to make up for the number of beats in the minute which have been lost, or unless there is also a great increase in arterial tension by contraction of the vessels. A very slow pulse depends in the great majority of cases upon a high arterial tension from vascular spasm—*i. e.*, resistance to the flow of blood; more rarely it is due to irritability of the vagus nerves, produced by pressure or disease, or by drugs, such as digitalis.

The term bradycardia is applied to a very slow pulse. The pulse may be as slow as twelve a minute.

A rapid pulse is seen most commonly as the result of stimulation

of the heart by drugs, by fever, or by fear. Fear causes the vagus to lose control of the heart, and fever acts by reason of the stimulant effect of heat upon this viscus. In other words, the quick pulse of fever is not a mere coincident symptom of fever, but the result of it. When the heart's action becomes exceedingly rapid it is called tachycardia. It is due in the majority of instances to relaxation of the bloodvessels, or more rarely to depression of the pneumogastric nerves. Often in this condition the pulse becomes so fast that it cannot be counted.

Great force of the pulse is due to hypertrophy, or over-action of the heart because of stimulation; and great feebleness generally is caused by marked dilatation not associated with hypertrophy, or in acute disease to exhaustion of the heart-muscle.

CHAPTER XI.

THE BLOOD.

The various forms of red and white corpuscles—Their proportionate number in health and disease—Alterations in their form and character—The hæmoglobin of the blood in health and disease—The various forms of anæmia—Leucocythæmia and pseudo-leucocythæmia—Parasites of the blood.

The Red and White Corpuscles. The condition of the blood, so far as alterations in it produce variations in the color of the skin, has already been mentioned in the chapter on that subject. We pass now to the study of the alterations which we find in this fluid, with the object of discovering what the changes appearing in it in disease may indicate. Before doing this, however, we must rehearse a few physiological facts in connection with the blood. The blood consists of a liquid basis or plasma, in which are found corpuscles, which can be divided into two great classes, the red and the white. The red appear as biconcave circular disks, dark at the edges and with a clear or lighter spot in the centre, due to their biconcavity. They do not contain a nucleus. They vary somewhat in size in pathological conditions, and when small are called "microcytes," when large "megalocytes," and when deformed or irregular in shape "poikilocytes." The red color of the blood is due to the aggregation of immense numbers of these bodies, the coloring-matter of which is called hæmoglobin; but if a few corpuscles are placed in a bright light on the stage of the microscope, they look bright and light yellow. The number of the red blood-corpuscles is about 5,000,000 in the cubic millimetre of blood of the adult male in health, and about 4,500,000 in the adult female. If the number is increased above normal, this condition is called polycythæmia; if decreased, the condition is called oligocythæmia. One of the most marked instances of polycythæmia which occurs is the very extraordinary increase of the red blood-corpuscles which is often seen in cases of congenital cardiac disease in childhood, amounting to as many as 8,000,000 to the cubic millimetre.

Not only may the number of red blood-corpuscles change, but the

quantity of their hæmoglobin may also vary. If the proportion is decreased, we call that condition one of oligochromæmia.

The white corpuscles, to which is often applied the term "leucocyte," appear in several quite distinct forms, which have nuclei; and, further, some of them possess the amœboid movement. The size of these bodies is very various, but they are larger than the red corpuscles as a rule. They are granular in appearance, do not arrange themselves in rouleaux, and, if the nuclei do not at first appear, these can be brought into view by the addition of a little acetic acid at the edge of the slide.

A few years ago little or no differentiation of these white cells had been made, but we now know that they occur in several very distinct forms, and that they are quite as important in aiding diagnosis as the red corpuscles. The simple examination of the white cells by the microscope is not sufficient, as they cannot be well distinguished one from another except by the use of certain stains which affect different corpuscles in various ways. Thus, one set of white cells, called "eosinophile cells" (eosine-loving cells), are called so because they are stained by eosine or by any acid stain, but not by other stains. Other cells are stained by basic stains, and still others by neutral stains. These are sometimes called basophiles and neutrophiles.

As will be still further pointed out, when speaking of the diagnosis of leukæmia from the appearance of the blood, the susceptibility of certain kinds of white blood-cells to certain stains is of considerable importance.

Five general varieties of these leucocytes are seen in the healthy blood of man: First, small lymphocytes containing one nucleus (an uninuclear or mononuclear cell), which nucleus is quite large and stains deeply when exposed to aniline dyes. They contain no stainable granules in their protoplasm. They form about 20 per cent. of the whole of the white cells in healthy blood. Second, large lymphocytes, which are also mononuclear, about twice the size of the small ones just named. The nucleus in these bodies is large and egg-shaped. Some believe that the small ones are the earlier forms of this class. Third, multinuclear leucocytes, the nuclei of which are irregular and subdivided. They are very granular in appearance (fine, dust-like granules), are strongly refractive, and are the most numerous of the white blood-corpuscles. They are called neutrophiles because they are stained with neutral stains, made by

mixing acid and basic colors. They are called sometimes polynuclear neutrophiles. They make up about 65 per cent. of the white cells in healthy blood. They are the corpuscles which are increased in the process called leucocytosis, or increase of leucocytes, in certain infectious diseases, notably pneumonia. Fourth. We find another class with very coarsely granular protoplasm, the nuclei of which have a great affinity for an aniline dye called eosine, so they are called "eosinophile corpuscles." One, two, or three nuclei may be found in cells of this class. Sometimes they are called "alpha corpuscles." In this corpuscle when stained by eosine the granules are purplish-red. The eosinophiles are very numerous in myelogenous leukæmia.

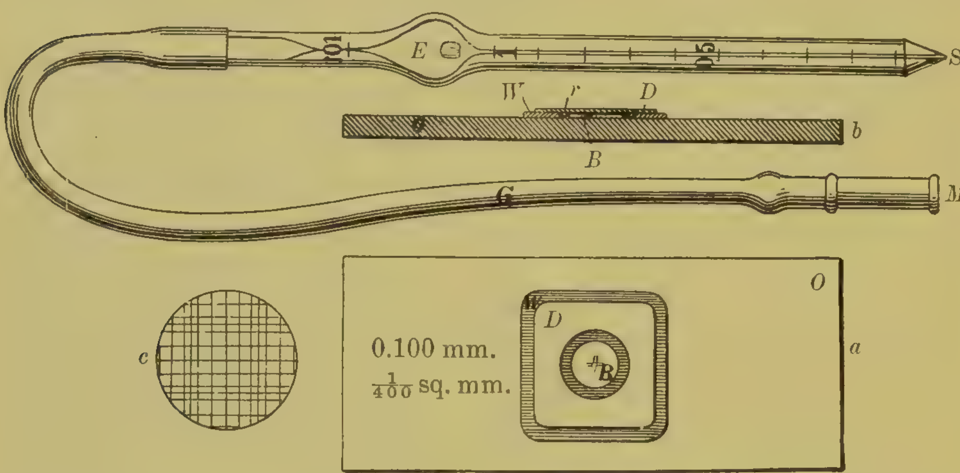
The proportion of white to red blood-corpuscles is as 1 to 500, but very great variations in the proportion occur even in health. Thus after meals the white corpuscles are always greatly increased, so that the proportion becomes as 1 to 100 or 1 to 150 of the red cells. On the other hand, after this primary increase following food they may become decreased, so that the proportion will be 1 to 800. Pregnancy and time of day are also factors producing variations in the proportion. Thus Hirt found before breakfast that the proportion was 1 to 716 ; one hour after breakfast 1 to 347 ; three hours after breakfast 1 to 1514 ; ten minutes after dinner 1 in 1592 ; half an hour after dinner 1 in 429 ; two and a half hours after dinner 1 in 1481 ; half an hour before supper 1 in 544 ; and 2 hours after supper 1 in 1227. In pregnancy the proportion was about 1 in 280.

We examine the blood not only by the microscope, but by color-tests. The object of the microscopic examination is both to determine the quality and character of the red and white corpuscles, their number, and the presence of parasites. The color-tests are for the purpose of determining the proportions of hæmoglobin, or, in other words, the ability of the corpuscles to carry oxygen to the tissues. To study the blood microscopically we need a quarter-inch objective for ordinary corpuscular work, or, as they say on the Continent of Europe, a Number 7 Hartnack or a D. Zeiss ; and for examinations for parasites a 1-12 oil immersion lens for use with a condenser. The eye-pieces used are usually Nos. 2 and 4.

The finger-tip of the patient having been washed clean, a sharp needle or the tip of a tenotome is used to puncture the skin and the drop of blood which escapes is placed upon a glass slide and covered with a cover-glass, so that the film of blood is very thin indeed.

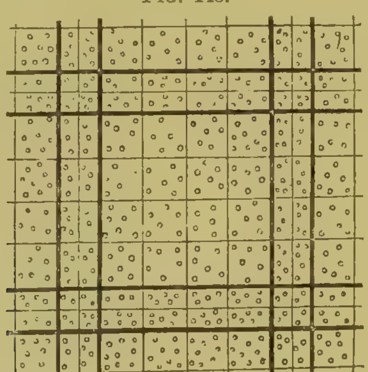
Examined under the microscope this will give a crude idea of the proportion of white to red corpuscles, and of their color and shape; but more accurate methods are advisable, and for their use we resort to what is called a hæmatocytometer, of which the best is the Thoma-Zeiss apparatus, which consists in part of a glass capillary tube,

FIG. 147.



Thoma-Zeiss blood-counting apparatus. A heavy glass slip (*a*), in the middle of which is a cell (*B*) exactly 1/10 millimetre in depth. The cell is limited at the periphery by a circular gutter to prevent fluid placed upon the cell from flowing beyond it between the slip and cover-glass. The floor of the cell is ruled into squares whose sides are 1/20 m.m. Dark lines mark out large squares containing twenty-five small squares. Thick, carefully ground cover-glasses (*D*) are provided in the case. The ordinary Potain *Melangeur* (*S*) is used to measure and mix the blood. It consists of a capillary tube the upper portion of which is blown into a chamber (*E*) holding 100 c.m.m. The stem of the tube is graduated at 0.5 and at 1 c. m.m.

FIG. 148.



Appearance of blood in the Thoma-Zeiss cell.

about 10 cm. long, with an expansion near the middle, which expansion contains a small glass ball which is movable. On the tube are three marks. Part way up it is marked 0.5, below the expansion 1, and above the expansion 101. The second piece of apparatus is a small cell 1-10 millimetre in depth, and the floor of

the cell is divided by finely drawn lines into squares. Each square equals 1-4000 cubic mm., and these squares are separated into groups of 16 by plainer lines. (Figs. 147 and 148.)

The finger having been freshly pricked, the blood is drawn up to the mark 0.5 in the capillary tube, and the tip of the tube is then wiped clean. A three per cent. solution of common salt is drawn up after it, until the tube and bulb are filled to the point marked 101. The tip of the tube is now wiped dry by means of a clean cloth. By shaking the glass ball in the tube the blood and salt solution become well mixed in the proportion of 1 to 200. After the salt solution in the lower part of the capillary tube has been forced out by gentle blowing into the upper end of the pipette and the blood-mixture has reached its tip, a drop of this homogeneous fluid is forced out into the cell just described and a cover-glass gently placed over it, all air being excluded. The cell should now be allowed to stand for several minutes to allow the corpuscles to settle and become stationary. The corpuscles in each of the sixteen squares in the cell are now counted, added together, and then the average number in a cell is obtained by dividing the number of corpuscles by the number of squares. This number in turn is multiplied by 800,000, and this result is the number of corpuscles in a cubic millimetre of blood; or multiply the number of corpuscles counted in all the squares by 4000 (4000 being the cubic contents overlying a square), and the result by 200 or 100, according to the dilution; after this divide the product by the number of squares, and the result will equal the number of cells in a cubic millimetre of blood.

If the blood is drawn up to the point marked 1 in the pipette before the saline solution is added, we multiply by 400,000 instead of 800,000, since the blood solution is twice as strong. In making the count it will be found that some of the corpuscles overlap the line of a given square, and may therefore be counted twice or left out altogether. For this reason it is customary to include these corpuscles which overlap the upper and left-hand borders. Further, it is best to put down the number of cells found in each square as they are counted and not to attempt to carry the addition in the memory, since the loss of one corpuscle makes a great difference in the ultimate result, and for this reason the more squares included in the original count the more accurate the result. For careful study of the blood several counts of several different fillings of the glass cell should be made and the result obtained by taking the average of these.

In making the count of the red blood-corpuscles care should also be taken to estimate the white corpuscles, since the proportion of white to red often gives us very valuable information in disease.

This may be done by using as a diluent for the blood in the pipette what is called Toison's solution, instead of the solution of salt already named. This has the advantage that it stains the white cells blue, and so renders them more readily counted. Toison's solution is composed of :

Methyl-violet	0.03	(½ grain)
Neutral glycerin	30.	(1 oz.)
Distilled water	80.	(2½ oz.)
Mix thoroughly and add		
Chloride of sodium	1.0	(15 grs.)
Sulphate of sodium	8.0	(2 drachms)
Distilled water	80.	(2½ oz.)

This is then filtered, and it requires about eleven minutes for the white corpuscles to be stained by it.

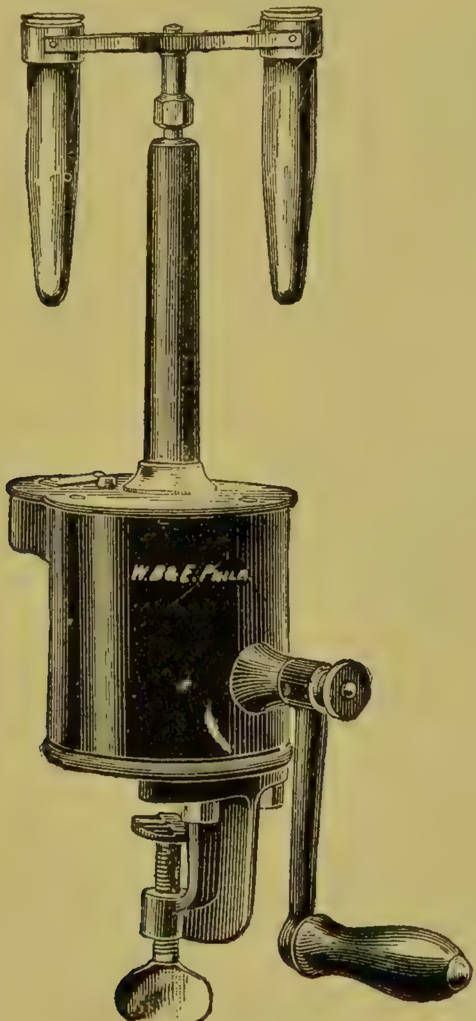
When we desire to count the white corpuscles alone we employ a pipette, which makes the dilution of the blood in the proportion of 1 to 10, and we use in place of salt solution as a diluent a 0.3 per cent. solution of glacial acetic acid in water. This acid solution dissolves the red corpuscles, but makes the white ones more readily seen. The method of calculating the number of white corpuscles in a cubic millimetre is the same as that given for the red corpuscles, except that as the dilution is 1 to 10 instead of 1 to 100 we multiply by 40,000 instead of 400,000.

Another very useful and rapid method of obtaining an approximate estimate of the number of the red and white blood-cells is by the use of the centrifuge, with the hæmatocrit attachment (Fig. 150) which takes the place of the tubes shown in Fig. 149.

This instrument is an improvement upon the Blitz-Hedin hematocrit, and is used for the volumetric estimation of the red and white blood-corpuscles without previous dilution of the blood. One turn of the handle of the centrifuge will rotate the upright shaft 65 times, and 77 rotations of the handle will cause the hæmatocrit to make 5000 revolutions per minute. This enormous speed is attained with only moderate exertion on the part of the operator. The hæmatocrit attachment consists of a metallic frame, carrying two graduated capillary glass tubes, 50 mm. long, ½ mm. bore, in which is placed the freshly drawn blood. These accurately graduated glass tubes, seated in rubber-cushioned cups at 1 and 2, are held in position securely by spring cups AA, so that there is no possible danger of

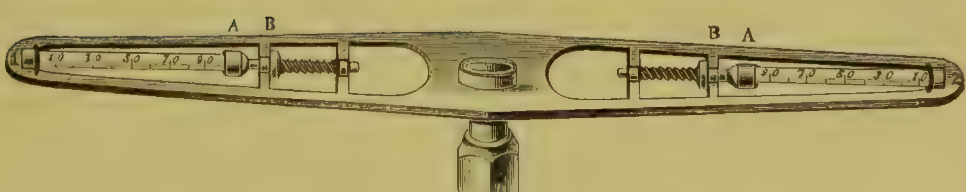
losing the tubes during rotation. By drawing back the milled heads BB the tubes are instantly released and as quickly clamped again

FIG. 149.



Centrifuge.

FIG. 150.



Hæmatocrit attachment for centrifuge.

into position. (Fig. 150.) This apparatus should be made of aluminum, in order that it may be strong and light. The advantage gained by the use of this metal is that it is possible greatly to increase

the length of the arms of the hæmatocrit; thereby taking advantage of the well-known law of mechanics that "that the centrifugal forces of two equal bodies, moving with equal velocity at different distances from centre, are inversely as their distance from centre." In order, therefore, to obtain any desired amount of centrifugal force it is not necessary to increase the speed of the machine but simply to increase the distance from the centre, or in the case of the centrifuge the length of the hæmatocrit.

The finger of the patient is thoroughly cleansed with water, and then punctured by means of a spear-pointed needle. The first drop of blood is rejected, and a second drop is secured by very slight pressure. The blood is then drawn, by suction, by means of a constricted dropper (Fig. 151) and rubber-bulb connection, devised by

FIG. 151.

Ashton and Stewart, and by this means placed in the hæmatocrit, which is rapidly revolved for at least one minute.

The rapidity and simplicity of this process are apparent at once. The blood does not have time to coagulate, and by the centrifugal force the red corpuscles having the greatest specific gravity are thrown to the distal extremity of the tube, and will occupy about one-half of the tube or to about the mark 50.

The white corpuscles, next in specific gravity, will occupy a position between the red corpuscles and the liquor sanguinis which is found in the proximal end of the tube, quite clear and free from corpuscles.

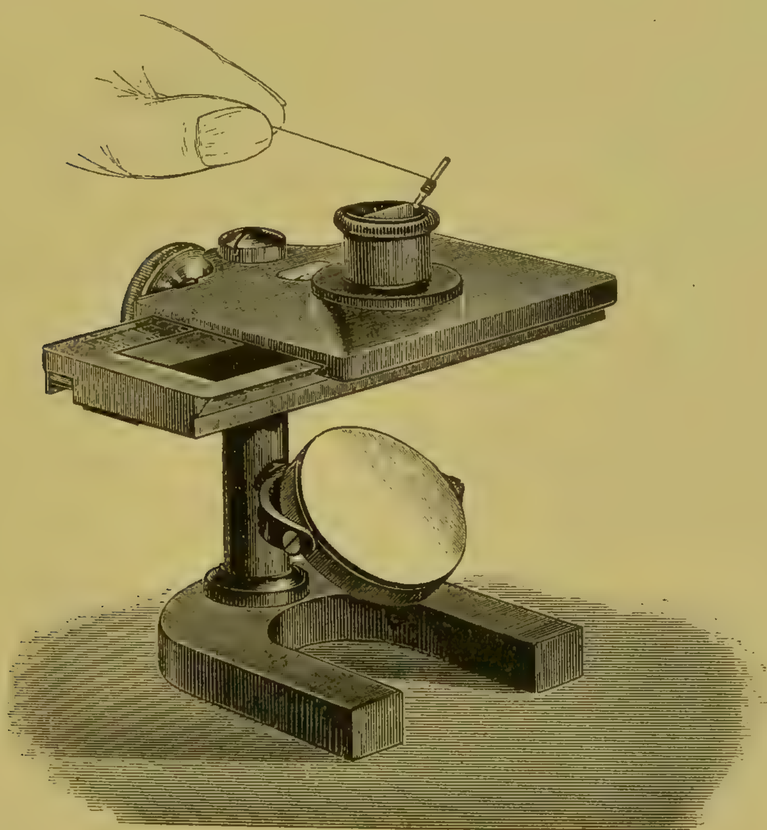
When the column of red blood-corpuscles extends to mark 50 we have, as a rule, about five million red corpuscles per cubic millimetre; but if the precipitated corpuscles reach only mark 30, there are only about three million per cubic millimetre, or sixty vol. per cent. If they reach only mark 20, there are about two million per cubic millimetre, or forty vol. per cent.

In examining the red corpuscles we should be on the lookout for defective corpuscles or ones which are deformed—that is, poikilocytes. We should also examine closely to discover the presence of nucleated red corpuscles, which, if present, are indicative of disease. These nucleated red cells have nuclei which are stained by the stains which

affect the white cell nuclei, and they occur in three forms, namely, as microblasts, normoblasts, and megaloblasts, or small, normal, and large cells. The microblast is rarely seen. The megaloblasts are commonly found in advanced leukæmia and in pernicious anæmia in its later stages. (See part of this chapter on Pernicious Anæmia.)

Having discovered the number and the quality of the corpuscles, an equally important measure is to discover the quantity of hæmoglobin which they contain. We do this best by the use of the hæmoglobinometer of v. Fleischl, although that of Gowers is sometimes

FIG. 152.



Von Fleischl's hæmometer.

used. Fleischl's apparatus consists in a small table, in the centre of which is a hole into which fits a round cylinder with a glass bottom, divided perpendicularly in the middle by a metal diaphragm, and both sides of which are filled with pure water. Under the stand is a frame in which is set a piece of colored glass as near the hue of diluted blood as possible, and this glass is tapered off gradually, so as to give a lighter shade of red at one end than the other. The frame carrying the glass is marked by a graduated scale and is moved

from side to side in a track under the half of the cylinder, which is to contain only pure water, by a thumb-screw. Under the glass and cylinder is a white reflector to direct the rays of light through them. (Fig. 152.) The rest of the apparatus consists in a little capillary tube attached to a tiny wire handle. This tube will hold just enough pure and healthy blood to color the water on one side of the cylinder to the hue shown in the colored glass when it is opposite the normal mark "100." The finger being punctured, the end of the capillary tube is lightly touched to the drop of blood which runs up the capillary tube, and the tube is then immersed in the water on one side of the cylinder, and stirred about till all the blood is washed out of it. The apparatus is then exposed to gas or lamplight, because with daylight the hue of the glass does not match blood-color, and the frame is moved backward and forward until the color of the glass, under the side of the cylinder which contains only pure water, seems to the eye to match the fluid containing the blood on the side through which the pure light streams. If the glass matches the blood-color when the mark on the frame is at 50, it shows that the hæmoglobin equals only 50 per cent. of normal, or if it is at 85, it signifies 85 per cent. As a matter of fact, an examination of perfectly healthy blood will often give not more than 85 to 90 per cent. of hæmoglobin with this apparatus.

Care should be taken in regard to three points : first, to be sure that all the blood is washed out of the capillary tube into the water ; second, to be sure that the two halves of the cylinder are filled to the brim with water so that there is neither a positive nor a negative meniscus ; and, third, to be careful to cleanse the entire apparatus thoroughly after each use of it before putting it away.

Anæmia. Having studied the methods of examining the blood we come next to the consideration of the diagnostic value of the conditions which we find in it. We find, first, that anæmia, or blood-deficiency, is represented by two conditions, in one of which the pallor and other symptoms are due to a diminution in the number of red blood-corpuscles, while in the other there is a decrease in the amount of hæmoglobin in each corpuscle. In regard to the white corpuscles we find even more valuable diagnostic data, since their variation in number, form, and character is marked in some diseases. Practically all conditions of the blood which are pathological represent diseases in organs connected with the blood directly or indirectly, and do not depend upon primary changes in this liquid, except in rare instances.

A patient's blood having been found lacking in the proper number of red blood-corpuscles, the question naturally arises as to what conditions underlie this variation from the normal. The most common causes of this decrease are the infectious diseases, which all result in producing a degree of anæmia most marked during early convalescence, and the history of such an attack should always be sought for, and, if found, regarded as an important point for consideration in reaching a diagnosis. If there be no history of acute illness, the most natural condition to be thought of is that known as simple anæmia, produced by no apparent disease of the organs of the body, but due to lack of good food, pure air, proper hygienic surroundings, and exercise. If this is excluded from the diagnosis, we must not forget that if food is taken and not absorbed properly the corpuscular richness of the blood is decreased, and therefore chronic indigestion, notably that condition called atrophy of the gastric tubules, may be the cause of the difficulty. Again, the presence of profound anæmia, as to the number of the red blood-corpuscles, may be present and seem inexplicable, until it is discovered that the patient suffers from bleeding hemorrhoids, and the daily loss of blood, even though it be small, is sufficient to produce anæmia. Similarly repeated attacks of nose-bleed or of excessive menstruation may so result. Naturally the physician will have excluded the possibility of the anæmia being due to a profuse hemorrhage from any cause before searching as far as this for a diagnosis.

There still remains to be considered the anæmia which is called "pernicious," in that it progressively gets worse till death occurs, in the majority of cases, although a few may recover. There is every reason to believe that in the near future we will understand its pathology, but at present we do not. It is characterized by marked pallor without loss of flesh, or, to speak more correctly, the subcutaneous tissues are added to rather than robbed of fat. There is gradual increasing dyspnœa, failure of strength, cardiac palpitation, venous murmurs, some vertigo, and roaring in the ears. The blood shows a most extraordinary and continually diminishing number of red blood-corpuscles, until the number may amount to only 143,000 to the cubic millimetre. In addition, the following points of great diagnostic importance are to be noted. First, the individual red corpuscles are richer than normal in hæmoglobin; second, many of them are larger than normal (megalocytes); third, the red corpuscles are deformed, some being

ovoid, others irregular in shape from projections and constrictions on their surfaces (poikilocytes); fourth, there are present microcytes or red blood-cells, which are smaller than normal; fifth, nucleated red blood-cells (normoblasts); and fifth, and quite constantly, there are other large cells like the megalocytes, named megaloblasts, which have a pale staining nucleus. These last are often larger than the megalocytes, and are sometimes called the "corpuscles of Ehrlich,"[1] since he regards them as pathognomonic of pernicious anæmia. The white blood-corpuscles are normal in number, or slightly decreased, although the great diminution in the red cells renders the proportion of white to red greater than normal.

Anæmia depending upon lack of hæmoglobin in the corpuscles, rather than a decrease in their actual number, is seen most typically in that condition called chlorosis. In this state the corpuscular diminution is so slight that it may be ignored; but the decrease in hæmoglobin is extraordinary, sometimes falling as low as 20 per cent. of the normal or below it. The red corpuscles are, however, very commonly irregular in form; that is, there is more or less poikilocytosis, but the white corpuscles remain normal in number or slightly increase. The diagnostic points, in addition to those of chlorosis just named, are the fact that the patient is generally a young girl of from fourteen to twenty-five years, that the skin is peculiar in its pallor (see chapter on Skin), and there is often little if any menstrual flow, which is usually only faintly pink in hue. Dyspnœa, cardiac irregularity, constipation, and a wayward appetite are often present. Auscultation of the neck on the right side over the jugular vein will reveal a peculiar murmur called a "humming-top" murmur. Febrile movement of slight degree may also be present.

In addition to these causes of anæmia we find anæmia due to a decrease in both the corpuscles and hæmoglobin. A large proportion of these cases have already been mentioned when speaking of the anæmias of convalescence and hemorrhage, but a far more important cause of this condition, yet one often overlooked, to the great regret of the physician in later years, is the possibility of the cause being tuberculosis. Still other causes of such anæmia are cancer, sarcoma, and renal disease, particularly gastric cancer, in which condition the blood may resemble that of pernicious anæmia, and gastric ulcer, in which the loss of corpuscles may also be extraordinary, even if no

[1] These are not to be confused with the myelocytes of Ehrlich.

hemorrhage occurs. Chronic lead-poisoning, arsenical poisoning, and uræmic poisoning may cause it, and it arises from the presence of numerous forms of parasites in the bowels, such as tapeworm, anchylostomum duodenale, and last, and by no means least, malarial infection, either as manifested by acute attacks, frequently repeated, or by slow poisoning with the development of cachexia. (See further on in this chapter.)

Leukæmia. There yet remain to be considered those conditions in which we find not only those states already described, but, in addition, marked alterations in the white blood-corpuscles as well as the red, alterations of such moment that they become the salient features of the blood when it is examined, and they are of great diagnostic importance. The points of importance in examining blood in regard to the white corpuscles are their number and their peculiarities and kinds. The discovery that the proportion of white to red corpuscles is far too great, varying from the normal (1 to 450, approximately), should cause the physician, first, to exclude all possibility of transient causes of variation by making an examination at various times of day, or by excluding the presence of acute infectious disease characterized by leucocytosis—that is, the presence of an unusually large number of white corpuscles. This can be done, not only by excluding the presence of an infection, but also by the fact that in leucocytosis in infectious diseases the increase is solely in the polynuclear neutrophile corpuscles. Thus it is well known that the taking of meals increases the white corpuscles during digestion, and that exercise and massage do the same thing, at least so far as the proportion in the peripheral vessels from which we draw the blood is concerned. If, however, these causes are excluded, and we find a patient of from twenty-five to forty years of age and a male (in the proportion of 2 to 1) pallid and puffy-looking, dyspnœic, and feeble, with a marked and constant increase in the proportion of his white corpuscles, what does it mean? It probably means that the patient is suffering from leukæmia (leucocythæmia) in one of its two forms, namely, spleno-medullary leukæmia or lymphatic leukæmia, of which the former is by far the most common.

When the disease leukæmia is present in its spleno-medullary form, the typical change, aside from the great increase in the number of the granular neutrophile white cells already described, is the presence of an additional cell, which is supposed to be derived from marrow, and is called a myelocyte, sometimes called the "myelocyte of

Ehrlich." They are large mononuclear cells with their protoplasm filled with fine neutrophile granular masses.

We have already described the white corpuscles of healthy blood in the beginning of this chapter. We can, therefore, pass directly to a discussion of the means to which we resort when we desire not only to examine the blood for the number of these cells, but also for the abnormal myelocyte just named.

The best solution for staining purposes is that of Ehrlich, which is called a triple stain. It is composed as follows: Saturated watery solution of orange "g," 125 cc; saturated hydro-alcoholic (20 per cent. of alcohol) solution of acid fuchsine, 125 cc. These ingredients having been mixed gradually and thoroughly shaken, the following constituents are added, the shaking being continued: Saturated watery solution of methyl-green, 125 cc; absolute alcohol, 75 cc.

This solution should stand for several weeks to allow of sedimentation; it improves with age; and when it is used the supernatant liquid is to be drawn off by a pipette in order to avoid the sediment. This stain acts in a few minutes.

Some cover-glasses having first been well cleansed by alcohol and water, the surface of one is touched to a drop of freshly drawn blood, and then another cover-glass pressed on its surface until the blood is evenly distributed. The glasses are then separated and allowed to dry. After they have dried they are still further hardened over an alcohol flame or on a hot stage made of sheet copper, and kept at 212° F. for fifteen minutes to two hours. After this they are placed in the staining-fluid for from one to four minutes, then washed in pure water, dried, and mounted in Canada balsam or cedar oil. The Canada balsam should not be prepared with chloroform, as it will decolorize the specimen. The glass is then ready for microscopic examination with $\frac{1}{12}$ oil-immersion lens. The nuclei of the eosinophile corpuscles will be stained a reddish hue, the neutrophile granules in the granular leucocytes will be stained purple, and the nuclei of the ordinary leucocytes bluish-green or blue. (Plate X.)

The other changes found in leukæmia are an increase of the eosinophile cells, but the polynuclear cells with fine dust-like neutrophile granules are about the normal number. The red blood-cells are greatly decreased in number, and a large number of nucleated red cells may be seen. The hæmoglobin is decreased. Cavafy states that the amœboid movements of the white cells are impaired. The proportion of one to three red cells is often seen. The additional

PLATE X.

FIG. 1.

Severe Anemia with Leucocytosis.

Dry preparation. Fixed with picric acid. Stained with hæmatoxlin Böhmer, x 300.

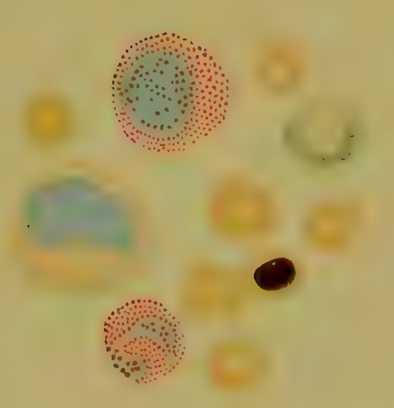
Red corpuscles few, almost colorless, varying in size, show poikilocytosis; two nucleated reds (normoblasts). The increase in the white cells seen to be in the polynuclear elements. (Rieder's "*Atlas der Klinischen Mikroskopie des Blutes.*")

FIG. 2.

Splenic-Myelogenic Leukæmia.

Eosin-hæmatoxylin, x 300. Red corpuscles rosy-red, of nearly uniform size, round. To the left a normoblast with eccentrically placed nucleus. Many large mononuclear leucocytes (myelocytes) and three eosinophiles seen. (Rieder.)

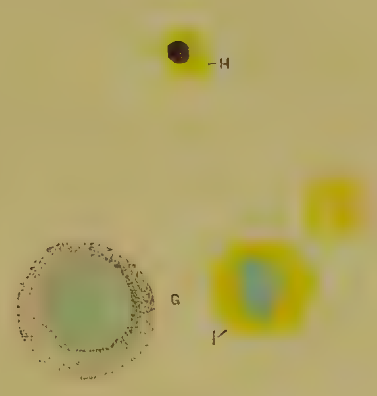
FIG. 3.



Splenic-Myelogenic Leukæmia.

Same case. Eosin-hæmatoylin, x 1100. One normoblast, one polynuclear leucocyte, one myelocyte, two eosinophiles. The neutrophilic granules of the polynuclear leucocyte and of the myelocyte do not show with this stain. The large mononuclear eosinophile above is believed to be also a myelocyte (Markzelle), the smaller one below, an eosinophile such as can be found in normal blood. (Rieder.)

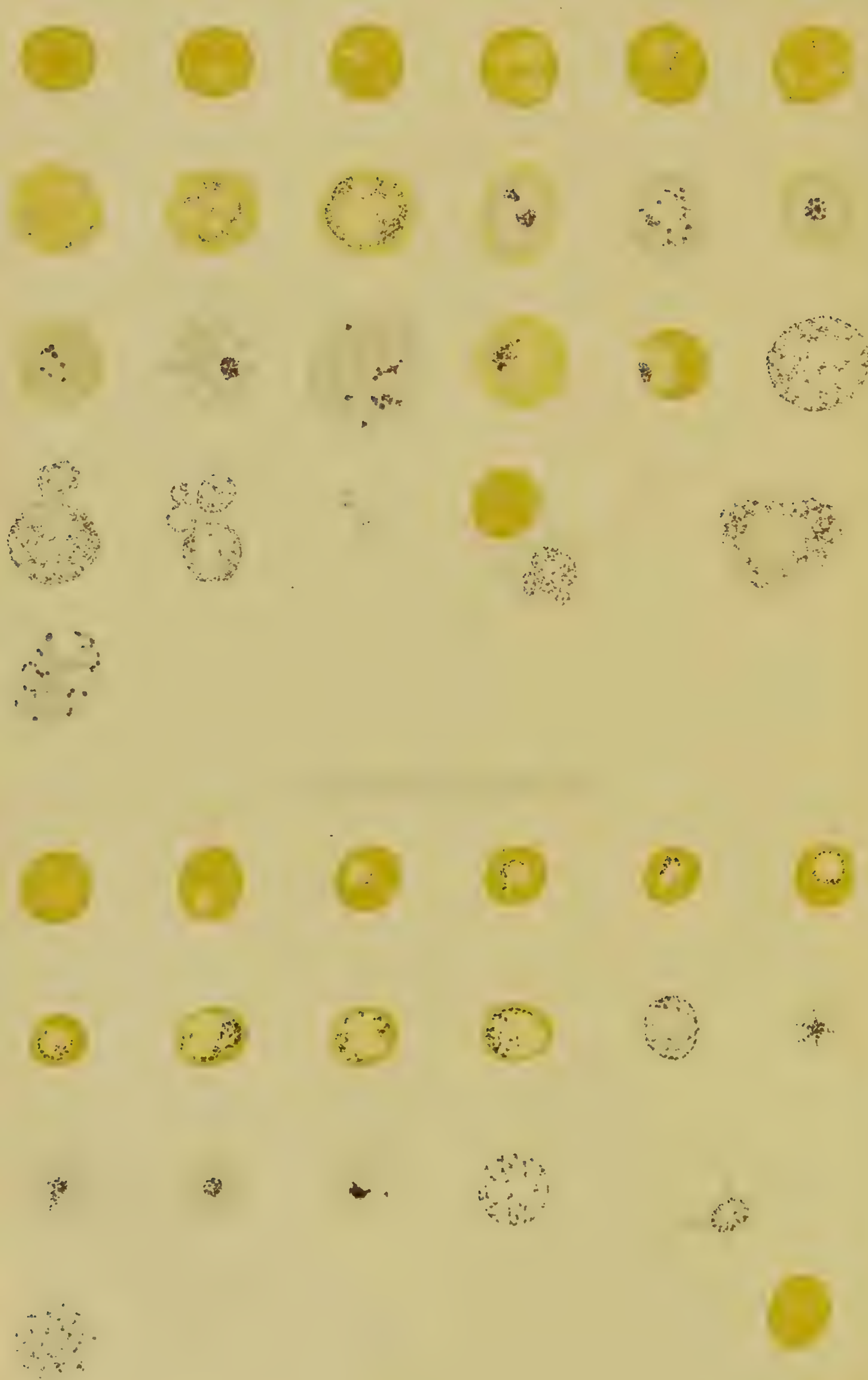
FIG. 4.



Myelocyte, Normoblast, Megaloblast.

Triple stain. G, myelocyte showing neutrophilic granules; H, normoblast, both from a case of splenic myelogenic leukæmia; I, large nucleated red corpuscle (megaloblast) from a case of pernicious anemia. (Osler.)

PLATE XI.



symptoms of this form of leukæmia are great and gradual enlargement of the spleen, with marked splenic tenderness. Auscultation over this organ may reveal a murmur and palpation a crepitus. Hemorrhage, generally from the nose, is common, and dyspnœa and diarrhœa are often present. Often retinitis develops, and slight fever may occur.

Lymphatic Leukæmia. When the proportion of white to red cells is one to ten, the white corpuscles all remaining normal in number, except the lymphocytes (that is, the mononuclear, deeply staining cells, with a rim of non-granular protoplasm), which are greatly increased in number, we suspect lymphatic leukæmia. Myelocytes, so typical of the splenio-medullary form, do not appear in this condition and splenic enlargement is absent, but in its place there is often enlargement of the superficial lymph-glands, but these never grow so large as in Hodgkin's disease or pseudo-leukæmia.

Parasites of the Blood. We still have for consideration the parasitic diseases of the blood. These consist in the malarial germ of Laveran, or, as it is more properly called, the "hæmatozoon malariæ" of Marchiafava and Celli, and the filaria sanguinis hominis.

Malarial Organisms. No more important addition to the study of disease, from a diagnostic standpoint, has been made than the discovery of the presence of a parasite in the blood of persons suffering from malarial fever, which is always present under these circumstances, and in all probability acts as the cause of all malarial manifestations. These parasites are varieties of sporozoa, which live inside the red blood-corpuscles of the individual attacked.[1]

The parasite of malarial fever occurs in three forms, namely, as that of tertian fever, that of quartan fever, and as the parasite of the so-called æstivo-autumnal fever. The tertian parasite is a small hyaline, colorless body, which occupies but a slight extent of the interior of the red blood-corpuscle. (Plate XI., Figs. 2, 3, 4.[2]) When quiet they are round like the corpuscle in which they lie; but if the specimen examined be fresh, they may be seen to possess active amœboid movements, thereby changing their shape.

Soon this amœboid body grows in size and begins to develop red-

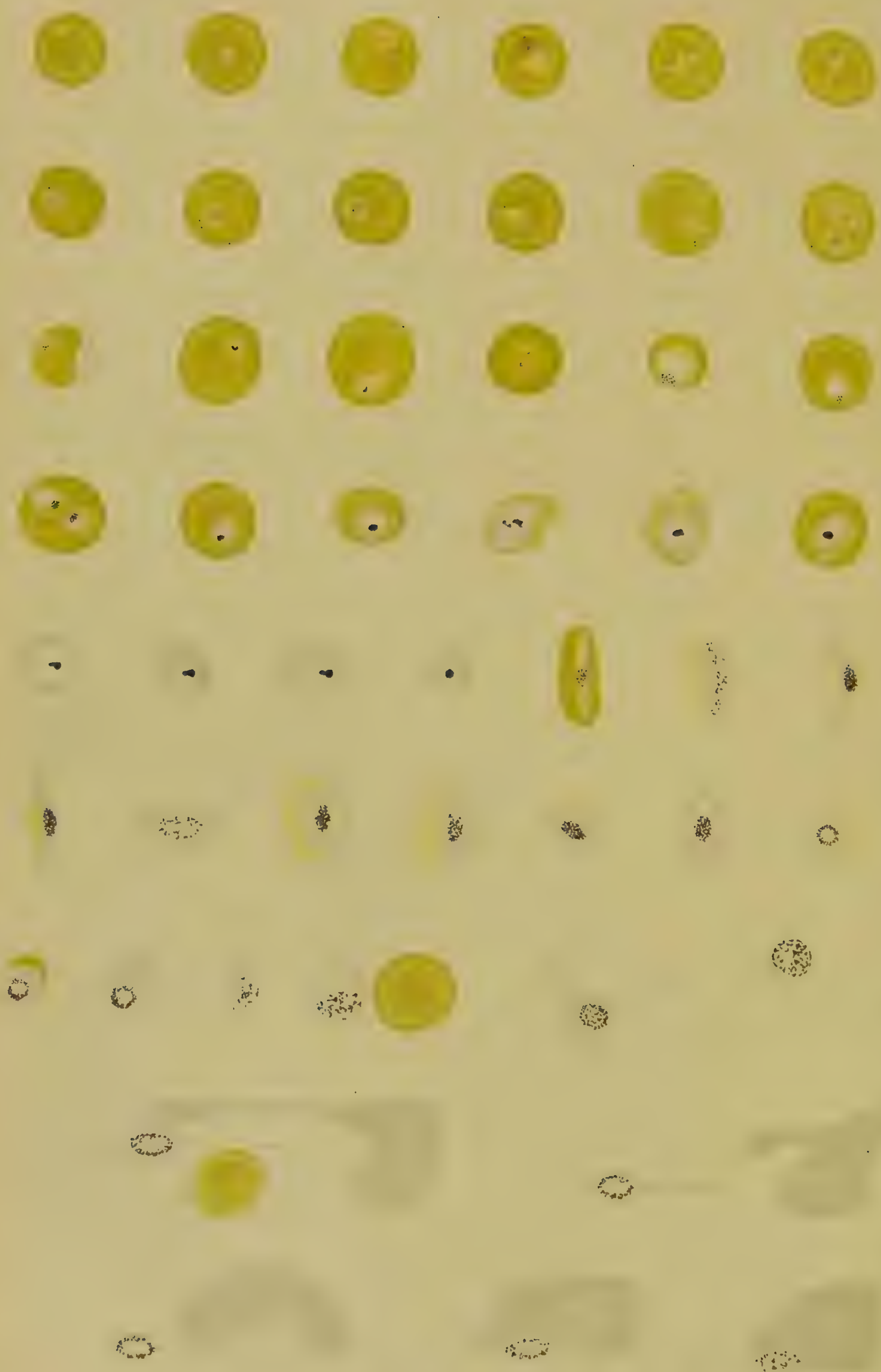
[1] In this country the chief investigators into the life-history of the malarial parasite have been Osler, Councilman, and, more recently, Thayer and Hewetson, from whose exhaustive and able monograph on "The Malarial Fevers of Baltimore" much of the information in the text of this book is derived.

[2] No. I. is normal red corpuscle. This plate is taken from Thayer and Hewetson's Nonograph.

dish-brown pigment-granules (Plate XI., Figs. 5, 6, 7) in itself. These pigment-granules are rapidly moving bodies, and as they are often found in the projections of the parasite, it may look, until this fact is corrected by fine focussing, as if several parasites were in one corpuscle. As the pigment-masses increase, the corpuscle which contains the parasite becomes more and more pale, and at the same time swells up or expands and the amœboid movements grow less and less, while the pigment tends to arrange itself toward the periphery. (See Plate XI., Figs. 7 and 8.) Finally, only a shell of corpuscle is left (Plate XI., Fig. 9), the pigment after collecting in the centre becomes motionless, and then the parasite undergoes segmentation; and, finally, we have developed 10 to 20 segments, arranged about the central clump of pigment like a rosette. Each segment has a spot looking like a nucleus, and soon the mature bodies so formed break out of their host and attack new and previously healthy blood-cells. Sometimes the parasite becomes so large that it entirely destroys the corpuscle and floats free in the blood, in which case the pigment-granules quiet down and the mass becomes misshapen and apparently dead, breaking up into smaller masses, and gives rise to several smaller bodies, which, however, soon seem to lose life (Plate XI., Fig. 21), or it becomes filled with vacuoles (Plate XI., Figs. 23, 24), or, finally, we have springing from these extra-cellular bodies flagella or waving arms, extending from the margin of the parasite. (Plate XI., Fig. 33.) These flagella break off now and again and keep waving through the blood, looking like spirilla. The entire process just described seems to consume about forty-eight hours, and it is of interest to note that the acme of the paroxysm of the disease occurs with the segmentation of the full-grown parasite, so that the presence of segmenting bodies indicates the near approach of an attack. If, on the other hand, we have a double tertian infection—that is, an attack daily, or a quotidian form, we have two sets of parasites, each one of which reaches its period of segmentation on alternate days, and so a daily attack is caused. In such blood during a paroxysm will be found two sets of parasites: one set segmenting or causing the paroxysm, and the other set half-developed, which produce the attack of the morrow.

The quartan parasite, or the one causing an attack every third day in its earlier stages of development, looks very much like that of the tertian form, for it occurs as small hyaline amœboid bodies filling a fraction of the corpuscle. They soon, however, develop

PLATE XII.



the following differences: first, they develop a sharper outline; second, they are more refractive; third, the amœboid movements are slower (Plate XI., Fig. 26); fourth, the pigment-granules are coarser and darker (Plate XI., Fig. 27), and, more important still, they lie very quietly around the edge of the parasite; fifth, the corpuscle acting as host does not increase in size, and finally disappear as it does when affected by the tertian type, but grows smaller and darker, more refractive and metallic-looking (Plate XI., Figs. 28 to 34). Reaching their complete development at about 64 to 72 hours, they appear as small, round bodies, taking up nearly all the space in the corpuscles in which they live, or they appear free in the blood-serum (Plate XI., Fig. 35). As the time for the paroxysm approaches the pigment-granules which have been scattered begin to collect at the centre (Plate XI., Figs. 36 to 39) in a stellate form, and the protoplasm of the mass then divides by segmentation into from six to twelve small pear-like bodies, each of which has a refractive centre. These bodies become more and more separated from one another, and simultaneously we find new corpuscles infected by the original small round bodies which we first saw.

Sometimes these parasites expand, become very transparent, and their pigment-granules become very active, but finally become quiet, and the body of the parasite grows more and more indistinct. They become dead parasites. (Plate XI., Fig. 40.)

Again, the parasite may undergo a breaking up into smaller bodies, which are badly formed and indistinct; also a degenerative form and vacuoles may develop. (Plate XI., Fig. 42.) Finally, flagella may develop, as in the tertian organism (Plate XI., Fig. 41), and they differ from the tertian form in being smaller, and their granules are coarser.

In the third form of infection (æstivo-autumnal fever) we find at first the small hyaline bodies, but they have a ringed appearance, and are sometimes very small. (Plate XII., Figs. 3 to 6.) Suddenly this body becomes larger and the ring is lost, the edge becoming wavy, and amœboid movements occur, the pseudopodia often joining to form a true ring. Pigment-granules finally develop after a variable length of time, but they are few, rarely more than two in a parasite, near the edge (Plate XII., Figs. 7 to 12), and quite still. The corpuscles are not decolorized, but often are shrivelled and very brassy-looking.

The peripheral circulation during the paroxysm of æstivo-au-

tumnal fever contains very few, if any, parasites, but blood drawn from the spleen may show intracorpuscular parasites, with blocks of pigment and some free parasites. As segmentation goes on, the parasite may look like the tertian form, but it is far smaller. (Plate XII., Figs. 21 to 28.) After this parasite has been present for some days we find in the blood larger parasites, of an egg-shape, or crescent shape, the remains of the blood-cell, looking like a small quarter of an apple glued to the side of the crescent. (Plate XII., Fig. 29.) Vacuolization and flagellation may develop in this form as in the others, and the use of quinine in the first week may prevent the development of the crescents.

The following table separates each of these malarial forms from the others :

Tertian Parasite.	*Quartan Parasite.*	*Æstivo-autumnal.*
Develops in 48 hours.	Develops in 72 hours.	Develops in 24 to 48 hours.
Pale and indistinct.	Sharply outlined and refractive.	Have a winged appearance.
Actively amœboid.	Slightly amœboid and later motionless.	Actively amœboid.
Pigment fine.	Pigment coarse.	Pigment-granules are very few.
Pigment active in movement.	Pigment slow in movement.	Pigment-granules quite still.
Pigment light.	Pigment dark.	
Full size of the corpuscle.	Smaller than the corpuscle.	
Degenerative forms twice as large as corpuscle.	Degenerative forms very much smaller than in tertian.	Very much smaller than a corpuscle.
Segments 16 to 20	Segments equal 6 to 12.	The process of segmentation goes on in the internal organs, so segmenting form is not found in the blood.
Irregular segments often.	Beautiful rosettes.	Forms crescents.
Corpuscle becomes colorless and swollen.	Corpuscle becomes brassy-looking and shrunken.	Corpuscle is shrivelled and very brassy, but not decolorized.

The blood is usually examined for the malarial parasite by what is called the direct or "without staining" method. The cover-glasses which are to be employed are cleansed very carefully by washing in alcohol and ether. The lobe of the ear, after being carefully cleansed, is then stabbed with a needle or small tenotome, and the first few drops of blood wiped away. A perfectly clean cover-glass is now picked up by means of a pair of forceps and touched to the tip of the drop of blood and then placed blood-side down upon a clean glass slide. The blood is equally distributed between the glasses, and only the merest touch of the cover-glass should be made to the drop of blood, as otherwise too much blood will be taken up.

The microscope should be fitted with a $\frac{1}{12}$ oil-immersion lens and a No. 4 eye-piece.

When it is desired to keep the specimen and to stain it, the best stain is that of methylene-blue and eosine, which is prepared as follows: A concentrated watery solution of methylene-blue is diluted one-half with water and mixed with an equal volume of a $\frac{1}{2}$ per cent. solution of eosine, in 60 per cent. of alcohol.

The blood is then obtained in the manner described above, but, instead of putting the cover-glass on a slide, two cover-glasses are placed face to face, and then after the blood is evenly distributed on each the preparation is fixed by immersing it for half an hour in absolute alcohol and allowing it to dry. This fixed cover-glass may now be dipped into the stain given for from half to one minute, and having been washed free from an excess of stain is ready for examination.

We can use another method, in which we stain by placing the dried cover-glass in Chenzynski's solution and gently heating it for fifteen minutes. This solution is made as follows: Methylene-blue in saturated watery solution, 40 c.c.; eosine in $\frac{1}{2}$ per cent. solution in 60 per cent. alcohol, 40 c.c.; distilled water, 40 c.c. The hæmatozoa are stained blue, the red cells and leucocytes light blue, their nuclei a deep blue, and the eosinophile granules of the cells deep red.

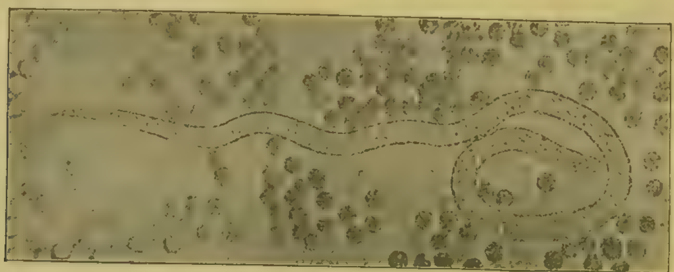
We have already stated that the paroxysm of the malarial disease takes place at the time when the parasite is breaking up into segments. In other words, the attacks occur whenever the cycle of growth of a set of parasites is completed, which in tertian fever is every forty-eight hours, and in quartan fever every seventy-two hours. If there be two sets of parasites in the blood, however, of the tertian type, the attacks may be daily, or quotidian, since each set mature on alternate days. This is often called double tertian. This is the most common form of the disease in the United States. If there be a double quartan infection, the attacks come on two successive days, then a day of intermission ensues. If three sets of parasites of this type are present, the attacks may be daily for three days, triple quartan infection. (See chapter on Fever.)

The parasite of æstivo-autumnal fever is irregular in its development, and is often the cause of the irregular malarial fever seen in the fall of the year. It yields less readily to quinine than the others.

Filaria. The filaria sanguinis hominis appears in the blood in three forms and has been well described by F. P. Henry, of Philadel-

phia, in a recent paper. These three forms are: 1. *Filaria diurna*; 2. *Filaria nocturna*; 3. *Filaria perstans*. These names are indicative of the habits of the animal, the *filaria diurna* being found in the super-

FIG. 153.



Filaria alive in the blood. Instantaneous photomicrograph. Four hundred diameters magnification. Four millimetres Zeiss apochromatic. (HENRY'S case.)

ficial vessels solely or chiefly during the day; the *filaria nocturna* solely or chiefly during the night; while the *filaria perstans* is con-

FIG. 154.

Filaria in the blood. Eight hundred diameters. (HENRY'S case.)

stantly present in the capillaries of the integument. The *filaria diurna* and the *filaria perstans* are confined, thus far, to the west

coast of Africa and adjoining districts; while the *filaria nocturna* is pandemic in the tropics and endemic in certain sections of the United States. The adults of *filaria nocturna* have been frequently found; that of *filaria perstans* never, so far as Henry has been able to ascertain. In the opinion of Manson, the *filaria loa* of the eye of the negro of Old Calabar is probably the adult form of the *filaria diurna.* If it is not, he argues, then there must be another blood-worm yet to be discovered, for the embryos of the *loa* must escape from the body of their host through the medium of the circulation. The *filaria perstans* has been practically proved by Manson to be the cause of the fatal "sleeping-sickness" of the Congo region.

The second is the one ordinarily seen in blood obtained from the peripheral circulation during sleep or at night. (Figs. 153 and 154.) The male filaria measures 83 millimetres long by 0.407 millimetre broad, and the tail is twisted into a spiral form. The female measures 155 millimetres long by 0.715 millimetre wide, and the vulva is 2.56 micromillimetres from the anterior extremity. The embryo measures 270 to 340 micromillimetres long by 7 to 11 micromillimetres wide, and has a pointed tail. This embryo is in an almost imperceptible shell which does not impede its movements, and as it is about the size of a red blood-corpuscle it passes through the capillaries in extraordinary numbers. Its active movements and typical appearance render it readily seen in the blood. The discovery of this parasite in the blood renders a diagnosis certain, and it should always be sought for if chyluria or elephantiasis is present. If the patient remains awake at night and sleeps during the daytime, the organism will be found in the blood during the sleeping-period.

The filaria diurna is found in the blood during waking-hours, and the embryos of the filaria perstans are the only form of this parasite known.

CHAPTER XII.

THE URINARY BLADDER AND THE URINE.

Disorders and diseases of the urinary bladder—Retention of urine—Incontinence of urine—The characteristics of normal and abnormal urine—The normal and abnormal contents of the urine—Their significance—Tests for the contents of the urine.

THE urinary secretion is one which is too frequently ignored by the student and physician in studying the diagnosis of disease. In many instances it will, if properly tested, give such positive evidence in regard to obscure affections that a correct diagnosis is at once possible, and in other cases its examination, as a matter of routine, will discover important facts the existence of which has been unsuspected. Again and again will a diagnosis prove erroneous if the importance of urinary examinations is ignored, and costly errors for the patient and the reputation of the physician ensue.

In asking questions about the character of the urine passed and its quantity, the physician should be sure that the patient clearly understands his questions. Often we will be told that much urine is passed, when, in reality, it is only in small amount, but passed often; or that it is blood-red, when simply red from urates and uric acid. In inquiring about its color, we should remember that if large amounts of liquid have been swallowed it will probably be light in hue, or, if small amounts of drink taken, dark in hue.

Anomalies connected with the urine may be divided into those which involve the organs which secrete, retain, and expel the fluid, and those which are manifested in the urine itself by alterations in its quantity, odor, specific gravity, and in its naked-eye appearance, its microscopical appearance, and, finally, by those changes which are discovered by means of tests which possess no influence of note on the urine of the healthy.

The objective symptoms of many cases of disease of the kidneys have already been discussed. (See chapter on the Skin, such as œdema and color of the skin, and chapter on Face, expression.) Aside from these evidences of renal disease no alteration can usually be noted unless it be loss of weight. The subjective symptoms of

the patient commonly consist in loss of ambition, malaise, disturbed digestion, and shortness of breath. Rarely is there pain in the lumbar region, unless pyelitis, stone in the kidney or ureter, or perinephritic troubles are present, when pain becomes an important sign.

FIG. 155.

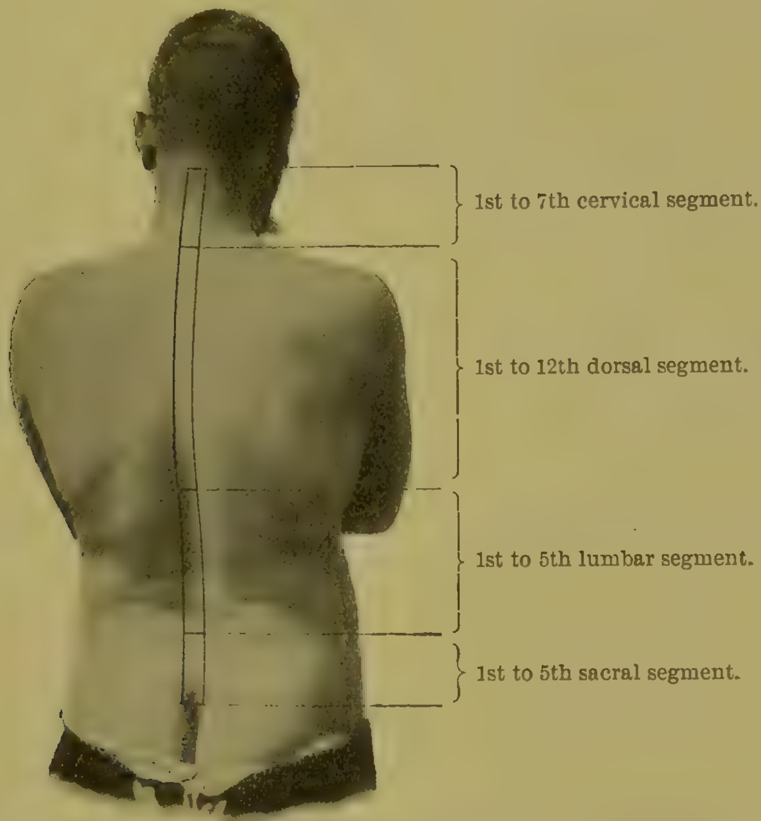


Diagram showing the surface-areas of the back corresponding approximately to the areas of the spinal cord supplying the trunk and limbs.

THE BLADDER. The objective symptoms of bladder-difficulties are generally local, unless they are very chronic, when the face may appear worn and weary, and, if a purulent cystitis be present, septic fever may occur. The subjective symptoms are tenderness, tenesmus, and pain (see chapter on Pain and chapter on Abdomen), and retention or incontinence of urine. Retention of urine, so far as the bladder itself is concerned, is rare, the cause of the retention generally being outside this viscus. It may, however, arise from disease or injury which destroys or temporarily impairs the function of the cells in the spinal cord which govern the contraction of the muscles involved in expelling urine from the bladder. These centres are situated at or about the level at which are given off the second, third, and fourth sacral nerves. (Fig. 155.)

Paralysis of the bladder with retention may, therefore, follow severe injuries to the spinal cord produced by falls, blows, or other traumatisms, or be due to a myelitis which destroys such centres. (See chapter on Legs and Feet, part on Paraplegia.) Again, retention of urine may arise from paralysis of the muscular part of the vesical walls by pressure produced in severe labor (childbirth).

Retention sometimes comes on in locomotor ataxia, in which disease the impulses from the bladder are not recognized, or are perverted so that the sphincter which closes the bladder does not relax to permit the escape of urine, or the cord or brain fails to recognize that the bladder is full, and so sends no impulse for its relief. Finally, we see cases in which the bladder cannot be emptied, because the walls of the bladder have been paralyzed by overdistention with urine.

On the other hand, incontinence results from loss of power in the sphincter, due to injury or disease in the cord at the level of the second, third, and fourth sacral nerves; and this, by the way, is a far more frequent occurrence than is absolute retention. The real condition under these circumstances is that the expelling-muscles and retention-muscles are both paralyzed, so that the urine accumulates in the bladder and then dribbles through the unguarded neck into the urethra. Sometimes, too, this incontinence is caused by the urethra being so insensitive that it fails to recognize the presence of the urine, and so does not send an impulse to the sphincter to tighten its hold. Incontinence also results from excessive reflex irritability of the walls of the bladder, so that the urine no sooner trickles into this viscus than an impulse is sent to the spinal centres which send a motor impulse to the muscles of expulsion. This is often the condition in the nocturnal incontinence of children, for as soon as the child sleeps its will-power over the bladder ceases, and reflex activity is alone in control. Irritating, concentrated urine may pervert the reflexes of the bladder and so cause incontinence.

The bladder-symptoms seen in myelitis—transverse, traumatic, or otherwise—usually come on in the acute form within a few hours after the sensory and motor disturbances have been noticed by the patient, and either incontinence or retention, or both, may occur.

If, however, the myelitis is not complete the bladder may escape. On the other hand, if the portion of the cord which is involved happens to be that part governing the bladder, vesical symptoms may develop before the motor symptoms are clearly marked. Again,

it is a noteworthy fact that when recovery takes place vesical control may be regained before any marked improvement can be found elsewhere. Often the loss of control over the bladder is such that the patient cannot voluntarily expel the urine and cannot retain it, and it dribbles away without his knowledge. Under such circumstances there is probably a myelitis involving the lower part of the dorsal cord and the upper and lower part of the lumbar cord; in other words, all that portion in which the vesical centres are situated. If the dribbling of urine takes place without distention of the bladder, the fluid passing directly from the ureters through the urethra, the lower part of the lumbar enlargement of the cord is affected, owing to the paralysis of the sphincter. On the other hand, distention of the bladder, due to retention of urine, occurs when the myelitis is in the lower dorsal and upper lumbar cord, and is due to paralysis of the detrusor muscles, which make no effort to expel the urine, while the sphincter, the centres of which are intact, maintains a tightly closed orifice. Such cases may empty the bladder spasmodically at long intervals (overflow-incontinence)—that is, sphincter-paralysis from distention may ensue. In such a condition the bladder should be emptied by the catheter to avoid paralysis and vesical disease. To put the case in another way, we can say that the spinal centre for the control of the walls of the bladder is situated at a higher point in the cord than is that for control of the sphincter, and, therefore, retention of urine indicates a lesion higher up in the cord than does incontinence without retention. Precisely similar vesical symptoms occur in cases of spinal tumor producing transverse lesions of the cord (see chapter on Feet and Legs, paraplegia), or may arise from spinal apoplexy.

The bladder-symptoms of locomotor ataxia are often quite characteristic, and are to be separated from those of myelitis, spinal tumor, and the vesical troubles due to traumatisms of the cord. The disorder depends entirely upon interference with the reflexes of the viscus, and so presents varying symptoms which are motor and sensory. The patient sometimes complains of the fact that he has to strain for a long time before he can start a stream which, even after it is started, is often jerking or interrupted; or, again, he must sit down and bend over in order to have the aid of his abdominal muscles before he can evacuate the bladder. As a result of this, residual urine in excess is always present, and cystitis or milder degrees of vesical irritability develop. In other instances, the

desire to urinate comes upon the patient so suddenly and forcibly that the urine is voided before he can, with his impaired gait, reach a place where he can pass it in a proper manner; or, on the other hand, it may be retained and can only be removed by a catheter. Still others find that urine escapes on laughing, coughing, or sneezing, owing to lack of complete control of the bladder and its sphincter; or, again, after many attempts to urinate, the patient gives up the effort, only to be humiliated by an involuntary passage of urine immediately upon his penis being withdrawn into his clothes.

These symptoms differ so materially from myelitis as to make a diagnosis as to their cause nearly always possible.

In obscure cases of ataxia the vesical symptoms may aid the diagnosis quite markedly; thus the presence of bladder-symptoms would confirm a diagnosis of ataxia as against pseudo-tabes, due to peripheral neuritis. Again, in myelitis the presence of vesical symptoms points to that disease, and excludes from the diagnosis such affections as poliomyelitis and lateral sclerosis, affections in which vesical paralysis rarely, if ever, occurs. Precisely similar vesical symptoms are sometimes seen in cases of general paralysis of the insane, but the delusions of grandeur or melancholia and other characteristic signs of this disease separate it at once from ataxia.

The sensory disturbance of the bladder will be found discussed in the chapter on Pain, but it is worth noting here that accompanying the symptoms already named as characteristic of locomotor ataxia vesical crises of spasm and pain frequently occur.

When there is pain in the bladder, made worse by the attempted act of micturition, and tenesmus, with darting pain into the urethra, there is probably present a cystitis; but the physician should remember that cystitis may be present with almost no painful manifestations, even when in its acute form. In other cases this condition arises from concentration of urine, which produces irritation of the viscus, such as is seen in cases of acute nephritis or renal congestion. In children this concentration of the urine is the most common cause of nocturnal urinary incontinence.

Involuntary passage of the urine sometimes occurs in idiots, in some cases of insanity, in attacks of apoplexy or any condition of abnormal unconsciousness, and sometimes in very severe infectious diseases, such, for example, as diphtheria. Oftentimes it results in children from irritation of the foreskin or vagina, or from rectal

irritation produced by seat-worms, since all these causes disturb the reflex activity of the spinal centres.

Interference with the passage of urine may also arise from two causes, which are surgical in character, namely, stone in the bladder and tumors of the bladder, which are often situated near its neck, and so produce obstruction. Finally, that most commonly met with cause of difficult micturition, enlargement of the prostate, is to be remembered.

Aside from these causes of interference with the passage of urine, we must not forget the possibility of its obstruction by stricture of the urethra, nor should the physician ignore the fact that some persons have "nervous bladders," which will not respond to an effort of the will if any person is near by, although the urine is instantly passed as soon as the patient is alone.

The Condition of the Urine itself is determined, first, by its general appearance, quantity, odor, specific gravity; second, by its microscopical appearance; and, third, by its chemical reactions and response to tests. Any changes in this fluid of an abnormal character are solely symptomatic, and point with more or less distinctness to disorders of bodily metabolism, disease or disorder of the kidneys, ureters, bladder, or urethra, and sometimes of the prostate, testicles, vagina, or uterus.

The urine which is to be tested should always be passed directly into the vessel in which it is brought to the physician, and this bottle should be scrupulously clean; or, if the urine is passed into any other vessel, care must be taken that it is perfectly clean. When it is thought that urethral disease may obscure the investigation, a catheter should be passed, all urine in the bladder drawn off, and then the catheter allowed to remain in place, so that the urine will trickle directly from the ureters to the catheter, and so to a receiving vessel. This is very important when the urine is voided involuntarily. If the condition of the bladder is bad, this viscus should be washed out by boric-acid injections, in order to prevent it from contaminating the urine which is to be tested.

The quantity of urine passed by a healthy adult varies from two to four pints in the twenty-four hours, according to the amount of liquid ingested, the freedom of perspiration, and the amount of exercise.

The significance of any great and constant increase in the amount of urine passed in a given case is multiple. Thus, we find it greatly increased in any disease of the diabetic centre, or of the liver, or

pancreas, which results in diabetes mellitus; in diabetes insipidus, in some cases of neurasthenia, and in some cases of hysteria. It is also increased in many cerebral lesions. Hypertrophy of the heart, *particularly if associated with chronic contracted kidney*, causes an increase in the urine; and, therefore, if a patient has to urinate frequently or has to arise at night to empty the bladder, we suspect this trouble if diabetes is excluded. The same result ensues if the heart and kidney are stimulated to increased effort by the action of drugs, such as digitalis, caffein, or alcohol. We also find an increase in urinary secretion, without its possessing any grave significance, in convalescence from such diseases as typhoid fever and pneumonia.

The quantity of the urine is diminished in cases in which the heart fails to do its proper amount of work, with resulting stasis of the blood in the kidney, and whenever any large amount of liquid is taken away from the body, as in diarrhœa. It is also decreased by fevers and by the sweats following febrile movement. Persistent vomiting also has a similar effect. Parenchymatous nephritis, both acute and chronic, greatly diminishes the urine, and in grave, fatal illnesses urinary suppression also takes place.

The odor of freshly passed urine is faint, but characteristic. What is often called a "urine odor" is really due to the development of ammonia in urine which has decomposed. The odor is altered by many drugs and foods, notably by copaiba, turpentine, eucalyptus, valerian, musk, asafœtida, and by asparagus, and diabetic urine possesses a heavy, sweet odor.

The specific gravity of the urine varies from 1005 to 1040 at 60° Fahr.; but a persistently low specific gravity indicates chronic contracted kidney if no dietetic cause can be found, while a persistently high specific gravity either shows concentration of the urine as the result of fever, or, if the urine is light in color, the cause is probably diabetes mellitus, the high specific gravity being due to the sugar which it contains.

The naked-eye appearance of the urine often gives very important information, if its clearness, opacity, and color are studied. Its clearness and color are modified by the presence of blood or other pigments derived from outside sources, such as the educts of carbolic acid or salicylic acid, of senna or hæmatoxylon, and bile, urobilin, and many other substances coming from inside sources. Many of these causes may render it opaque, but there is one condition, above all others, which renders the urine cloudy even when freshly

passed, namely, cystitis with phosphaturia. After urine has stood for some hours and undergone chemical changes, it often becomes opaque.

When urine is dark red in color and somewhat opaque this discoloration may be due to blood, hæmoglobin, santonin, rhubarb, senna, logwood, and the presence of an excess of urates. Again, it may be rendered almost black, instead of red, by an excess of biliary coloring-matter, and a black urine is often seen in cases of melanotic cancer, the color being due to melanin.

If the color be due to blood or *hæmaturia*, the urine will be of a more or less bright red, according to the freshness of the sample brought to the physician and the seat of the hemorrhage. If the urine has been voided several hours, it will be of a dingy red or smoky hue, and on standing will cause a coffee-ground or reddish sediment of a somewhat flocculent appearance. If, on the other hand, the urine is seen as soon as passed, it may be a bright red or a dingy red, according to the seat of the hemorrhage and the time which has elapsed since the bleeding began; if it has arisen in the kidney or ureter or bladder, and has been gradual, the mixture of blood and urine will have been so intimate that changes in the blood will have taken place, whereas if the hemorrhage has occurred, simultaneously with urination, from the neck of the bladder or the urethra, the blood will be almost unchanged when it escapes from the urethra. The presence of clots in recently passed urine indicates a not very recent hemorrhage, and yet one of such size that the urine could not by dilution completely prevent clotting.

Blood from the kidney usually possesses the following characteristics: It is well mixed with the urine, and is generally altered in appearance to the naked eye and under the microscope, both as to color and the shape of the corpuscles. The cells and casts which may be present are changed in color by the hæmoglobin which is free in the urine. Again, blood-casts or red blood-corpuscles clinging to casts indicate renal hemorrhage. When the blood comes from the kidney pelvis it may appear in the urine in long, worm-like clots (moulds of the ureter), and their extrusion from the ureter produces symptoms of colic. Under such circumstances there may be alternations of hæmaturia and normal urine, due to the blocking of the ureter on the diseased side by a clot, so that all the urine comes from the healthy kidney. A sudden profuse hemorrhage in the urine, sufficiently large to endanger life, may come from cystic tumor of the kidney.

When the blood comes from the bladder it is generally due to some capillary growth or to injury. Rarely in certain cases of locomotor ataxia, hæmaturia develops after the vesical crises which we have already described (see Bladder in this chapter). The origin is capillary hemorrhage from the bladder-walls.

When the blood comes in the first part of the urine passed and not in the last part, it almost certainly comes from the urethra. The urine, when not discolored by blood, may be discolored by the presence of the coloring-matter of the blood. This is called hæmoglobinuria. Microscopical examination of the urine in such cases will show no corpuscles, although the urine will be coagulated by the acid test; but this coagulation does not settle in flakes as it usually does in albuminous urine, but floats on the surface in a brownish mass. The naked-eye appearance of the urine is that of clear port wine. If a few drops of urine be placed on a watch-glass, and a drop of strong acetic acid be added, the blood-crystals of Teichmann will be found, showing that the coloring-matter is hæmoglobin.

If the discoloration of the urine be due to blood, a microscopical examination will reveal red blood-corpuscles, white blood-corpuscles, and perhaps fine filaments of clots; but the corpuscles will not be found in rouleaux, as in ordinary blood outside the body, and they may be crenated and distorted in shape, particularly if the urine is alkaline.

The test which can be most easily applied to determine the presence of blood, if the microscope cannot be used, is Heller's test, which consists in adding to a few c.c. of urine a little caustic soda, so as to render the liquid strongly alkaline. The urine is now heated to boiling, and if blood is present a bottle-green color is produced, and the phosphates fall to the bottom of the test-tube in fine flakes, tinged brownish-red by the coloring-matter of the blood.

The significance of hæmaturia is various, since any solution of continuity in the bloodvessels of the genito-urinary tract may produce it. When the blood comes from the kidney some of the possible causes are acute parenchymatous nephritis, resulting from any one of the severe infectious diseases, such as scarlet fever or malarial fever; from embolism, resulting from ulcerative or other forms of endocarditis; renal infarction, from sepsis of the kidney; from the ingestion of irritating drugs, such as cantharides or turpentine; and from strains or blows of the back, producing rupture or other disorganization of the kidney. All these conditions produce what may

be called acute hæmaturia. If the cause be acute nephritis from the presence of an infectious malady, such as scarlet fever, the pain in the loins, the presence of albumin in the urine, and the eruption will render the diagnosis easy.

Hæmaturia due to malarial poisoning may appear with the first malarial paroxysm, of the intermittent type, which the patient has ever had, and at a time when the history of the case renders it certain that a hidden malarial condition could not have previously damaged the renal tissues or those of other organs in the body. In other words, there are cases in which a free hemorrhage from the kidney takes place, by reason of the chill, in much the same manner in which hemorrhage takes place in an acute nephritis due to exposure to cold or to irritants. Under these circumstances there may or may not be developed a true organic lesion of the kidney in the sense of permanent disease.

Secondly, we have cases in which bloody urine appears, not in the first malarial paroxysm of the intermittent type, but in association with the later attacks, which may have followed the first either rapidly or slowly. In these cases there may be no further cause for the hemorrhage than excessive congestion, but in all probability the vast majority of such patients present distinct renal changes, which permit such a symptom to develop when the paroxysm asserts itself.

Thirdly, we pass from those cases of bloody urine due to intermittent forms to those due to remittent attacks, which, in many cases, have gradually merged from the first into the remittent. In these patients the process by which a bloody-colored urine is developed may be very complicated, since it may be due to renal incontinence, functional or organic, or to a true hæmoglobinuria, arising from dissolution of the red blood-cells in the bloodvessels or blood-making organs.

Finally, there is a type of malarial hæmaturia which is only produced by the administration of quinine (Karamitsas *et al*).

All these forms of hæmaturia can be diagnosed by the presence of the malarial germ in the blood (see Blood) and the characteristic malarial symptoms, except that which occurs in persons who have a dyscrasia from old malarial poisoning.

If the hæmaturia be due to embolic infarction of the kidney, an examination of the heart will probably reveal signs of valvular disease, from which source the embolism will have resulted, or, in

other cases, the physical signs, combined with the history, will show malignant endocarditis with renal sepsis therefrom. If such disease is not present, the history of the ingestion of an irritating drug will be the diagnostic guide, or, if injuries be the cause, a history of traumatism is all that is needed to elucidate the case.

The causes of chronic or persistent hemorrhage from the kidney are chronic hemorrhagic nephritis, cancer of the kidney, calculus in the pelvis of the kidney producing ulceration, injury of the kidney by jarring of a stone, tuberculosis of the kidneys, and cystic degeneration.

If the chronic hæmaturia arise from chronic hemorrhagic nephritis, the diagnosis is made by the pallor of the skin, anorexia, nausea, headache, œdema, decreased amount of urine, and albuminuria.

If the cause be renal cancer, the cachexia, pain, and the mixture of pus, blood, and disorganized renal tissue in the urine will render the diagnosis possible. If due to calculus, there may be a previous history of attacks of renal colic or of violent pain in the kidney; and if ulceration of the renal pelvis has occurred, there will be disturbances of the body-temperature, pain in the lumbar area, and pus in the urine. The presence of tubercle bacilli in the urine decides the presence of renal tuberculosis. If cystic degeneration is present, it can only be determined if the cyst is large enough to be felt.

FIG. 156.

Distoma hæmatobium male and female. The two small bodies (10 diameters) are the eggs (150 diameters).

There are other forms of hæmaturia which must not be forgotten, although comparatively rare, namely, that due to the presence in the blood of the filaria sanguinis hominis, which is a condition in which the presence of chylous urine so overshadows that of blood that the appearance is that of pinkish cream or milk, and microscopical examination will show blood-corpuscles and fat-globules, as well as the embyros of the filaria. (See Chyluria in this chapter.) The second still more rare cause of hæmaturia is the distoma hæmatobium of Egypt and Abyssinia. (Fig. 156.) These produce what has been called tropical hæmaturia. The third cause is even

more rare in man, namely, the strongylus gigas, which also causes pyelitis and renal colic. A fourth form of hæmaturia is that seen in some cases of scurvy, and, lastly, hæmaturia may also appear as a symptom of purpura hemorrhagica, hemophilia, and very rarely in leukæmia.

Hæmoglobinuria arises from a number of cases, such as infectious disease, poisoning by mushrooms and certain coal-tar derivatives, chlorate of potassium, and glycerin. Malarial poisoning sometimes causes it instead of hæmaturia. One form of malarial hæmoglobinuria is intermittent, the urine being at one hour limpid, the next hour bloody, and the third hour clear again.

The possibility of confusing the hæmoglobinuria of idiosyncrasy about to be described, when in a severe form, with true and severe malarial poisoning is very great. The entire history of paroxysmal hæmoglobinuria teems with reports of cases in which the chief manifestations of a malarial attack were present, such as chills, fever, and sweats. Lichtheim and Ponfick have shown that the injection of lamb's blood into the vessels of man results in violent shivering, fever, sweats, and pain in the lumbar region over the kidneys.

This condition also follows severe burns and the transfusion of blood, and occurs in paroxysmal hæmoglobinuria, a condition which seems to be produced by mere chilling of the surface of the body or to immersing the hands of a susceptible person in iced water. It may also be produced either by exposure to cold and damp, which are generally present in malarial localities, or to the chill of the milder forms of malarial paroxysm.

If the urine be red from other caues than blood, it may be due to the ingestion of logwood. The history of the ingestion of this substance will clear up the diagnosis. If it be due to senna, it will be carmine, due to the chrysophan in this drug ; but this only appears if the urine is alkaline. Precisely similar changes are due to the taking of rhubarb. So in santonin-poisoning a blood-red urine is sometimes seen, but it usually attains this appearance after being at first yellow, then saffron, and then purple-red. One of the conditions of the urine, due to a poison which can be readily confused with hæmoglobinuria or hæmaturia, is that produced by carbolic acid. This color is not due to blood, but to oxidized educts of the acid. The same educts produce a similar discoloration after naphthalin, creosote, and uva ursi have been taken in overdose.

Finally, the urine is often dark reddish-brown or porter-colored

in jaundice, owing to the presence in it of biliary coloring-matters. Under these circumstances it may be clear or opaque, and the fluid is apt to be frothy on shaking and to have an increased surface-tension, so that powdered sulphur does not sink to the bottom of the vessel when the sulphur is dropped on the urine. These biliary colors are at once recognized by the reaction with nitric acid in Gmelin's test, for, if a little of the urine be placed on a white plate and nitric acid be allowed to touch the margin of the wet place, the play of colors from green to blue, blue to violet, and violet to red occurs. The green color is the only one characteristic of the biliary reaction, for indican will give with nitric acid the other colors. The same test can be used by wetting bibulous paper with urine, and the acid, if brought to the edge, will stain the paper in the colors named. (For the symptoms of jaundice, see the chapter on the Skin.)

A greenish-colored urine is seen in cases of poisoning by salicylic acid, due to the indican and pyrocatechin; and from the use of saffron. The urine is yellow in santonin-poisoning, and when rhubarb has been taken, if it is alkaline.

When through diseased processes indican is formed and excreted in the urine, it may be by oxidation transformed into a blue color (indigolin),or into a red hue (indirubin). If chromogen is present in large amount, shaking the urine with air will develop a violet-blue color, or this change may take place in the bladder. If urine containing indican be treated with two or three times its volume of hydrochloric acid, it will turn a violet hue.

Indicanuria is present in intestinal obstruction, general peritonitis, cholera, cancer of the liver or stomach, and pernicious anæmia. It may, however, be present in health as a result of constipation. Blue urine is also caused by the ingestion of methyl-violet as a drug.

A black urine is sometimes seen in a case of melanotic cancer, or after the brownish urine produced by carbolic acid or uva ursi has been exposed to the air.

Red urine, due to none of the causes which have been enumerated, may be due to an excess of urates (except urate of sodium, which is usually white). If the urine becomes brown on the addition of nitric acid when the fluids join, it is due to urates; but if all the fluid is brown, the patient has probably been taking iodine or iodine compounds freely.

White or milky-looking urine is seen in that condition called

chyluria, due to the presence of filaria sanguinis hominis in the blood. This urine on standing forms a creamy layer on its surface, and, if it is shaken with ether, some of the fat can be removed, rendering the urine clear. The diagnosis can only be confused by urine becoming mixed with milk or cream, and can always be made if the embryos of the filaria can be found in the urine. They lie in very delicate sheaths, and show a constant vibratory movement. The diagnosis is still further confirmed if they are found in the blood, where they are present in large numbers at night. (See chapter on the Blood.)

Urine may have a somewhat milky-white appearance from an excess of phosphates, mixed more or less with mucus, as in catarrh of the bladder.

When the urine is passed in large quantities, and is of a very pale straw-color or has a slightly greenish tinge, it will often contain sugar; or, in other words, be the urine of diabetes mellitus, or of glycosuria from other causes. The fact, that it remains markedly acid for a long time after it is passed, and that it has a high specific gravity, point still more to its being diabetic, and the diagnosis is confirmed if the characteristic reaction with Haines's, Whitney's, or Fehling's solution is obtained. (See Tests in this chapter.)

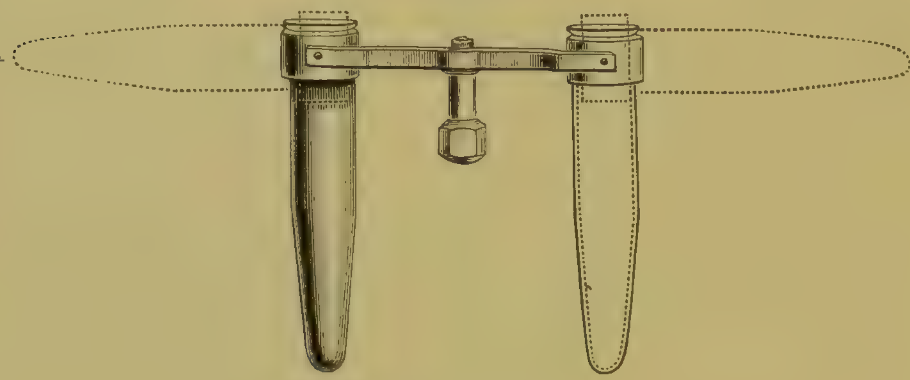
Microscopic Appearances of the Urine and its Contents. Having considered the macroscopical appearance of the urine, we may turn to its microscopical appearance, and this part of the subject is of even greater importance than the study of the gross appearance of this secretion, for, very commonly, a sample of urine which looks quite normal to the naked eye is loaded with microscopic objects of the greatest pathological significance. The most important of these objects are what are called "casts"—that is, moulds of the uriniferous tubules, formed as a result of the disease-process present in the kidney.

These casts consist of epithelial cells, blood- and pus-corpuscles, masses of micro-organisms, or of broken-down organic matter, as in fatty casts, and in hyaline or transparent bodies, or moulds which are made up of unknown material, but often covered by corpuscles, pus-corpuscles, or epithelial cells. In addition to these bodies we find a large number of organic bodies or derivatives of organic matter, and inorganic substances derived from the tissues or from food.

The reader who desires to examine urine successfully by the aid of the microscope must bear in mind that it can only be examined

satisfactorily after it has stood still in a glass or other vessel for a long enough time to allow sedimentation to take place—that is, until the objects floating in the fluid have had time to settle.

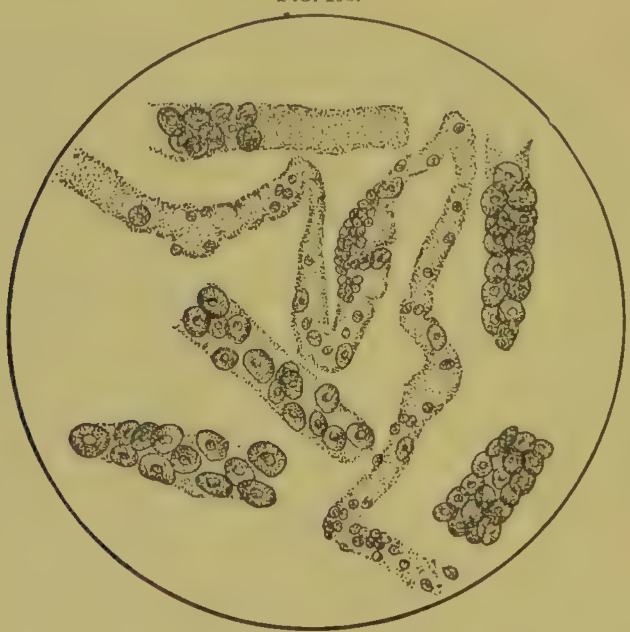
FIG. 157.



Holder for urine-tube.

By far the best method of obtaining the sediment, however, is by the use of the centrifuge, an apparatus by means of which the solids in a fluid are separated by centrifugal force. In this apparatus a

FIG. 158.



Casts containing epithelial cells. (PEYER.)

sediment can be obtained in a few minutes after urine is passed. (Fig. 157 and chapter on Blood.)

This sediment is to be drawn up into a pipette which has been introduced into the urine and a few drops placed upon a glass-slide,

after which the drops are to be covered by a cover-glass and the slide placed under the microscope.

Casts composed of epithelial cells present an appearance similar to that seen in Fig. 159, and are due to proliferation or exfoliation of the epithelium lining the uriniferous tubules. The cells look swollen and granular and may contain globules of fat. These epithelial casts occur in three forms: first, they may appear as hollow casts of the tubule when the epithelium has exfoliated *en masse* (that is, the lining of the tube is cast off in one piece); second, they appear as casts made up of epithelial cells glued to one another; and, third, the cells are attached to the surface of a clear transparent basis, looking like a hyaline cast. All these varieties are highly refractive of light and are not altered by chemical substances as easily as are the other casts about to be described.

Having found bodies of this sort in the urinary sediment, what is their significance? They are a positive sign of an inflammatory process in the parenchyma of the kidney, or, in other words, of parenchymatous nephritis.

Blood-casts consist of more or less well-preserved blood-corpuscles, attached to one another in a mould of the tube in which they have escaped. They are rarely seen and are masked by freely floating cells. The significance of these blood-casts is great, as they indicate an acute inflammation of the kidney, acute congestion of this organ, or a renal infarction. They are of importance, too, in separating hæmaturia arising from other sources than the kidney from hemorrhage of this organ, because they are not found unless the escape of blood has been in the uriniferous tubules.

Casts composed of pus-corpuscles are still more rarely seen, but, if constantly present, may indicate multiple abscess of the kidneys.

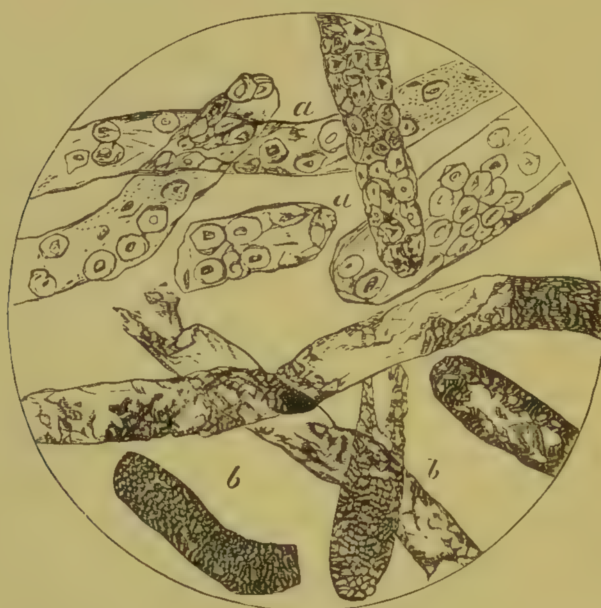
When masses of micrococci become grouped together in the tubules they may be expelled in casts, and under a low power look somewhat like granular casts (see below). They can be seen to consist of micrococci if a high power is used, and they are not quickly changed by acids, as are casts composed of other materials.

The significance of their discovery is that septic infection of the kidney is present, as the result, it may be, of septic embolus brought from a distant infected part. They are seen in suppurative renal inflammation and in cases of pyelonephritis in which the true renal tissues are being involved by an extension of the disease.

Casts, composed of broken-down organic matter, are found as

granular and fatty bodies; that is, they represent broken-down blood-corpuscles and epithelial cells, and their appearance varies greatly according to the stage of the process and the origin of the materials composing them. Thus, the granular appearance may be very fine, as shown in Fig. 159, or light and refractive, dark or opaque. Very often the edges of these casts are irregular and the ends frayed and uneven. The color of these bodies may be yellow, brown, or grayish.

FIG. 159.



a a. Epithelial casts. *b b.* Opaque granular casts, from a case of acute Bright's disease. (ROBERTS.)

The significance of granular casts is not as positive as those named so far, but they often indicate a slow degenerative process in the renal parenchyma.

Fatty casts, composed of minute globules of oil, cohering to one another or attached to a central core of epithelium or fat-crystals, are found in cases of widespread fatty degeneration, as the result of disease or poisoning, as in the case of large white kidney on the one hand, or phosphorus, arsenical, antimonial, or iodoform-poisoning on the other. They show the presence of a very slow process if due to disease, but have not the same significance if caused by poison. (Plate XIII., Fig. 1.)

Hyaline casts are long, worm-like, transparent bodies, with very fine granulation, particularly along the edges, and because they are transparent they are often hard to find. These bodies are supposed to

be composed of albumin which has been exuded into the tubules. Their significance is exceedingly grave, as they point very strongly to that incurable malady, chronic interstitial nephritis. If the casts are very large, they may show amyloid degeneration of the kidney. They have often been wrongly called "waxy" casts. (Plate XIII., Fig. 1.)

Casts are not to be confused with cylindroids or streamers. These cylindroids appear in several forms. Most commonly they look like threads or filaments which are transparent and often somewhat striated or hyaline in appearance. They are often long enough

FIG. 160.

Albuminous urine. *a*, *b*, and *c*. Ribbon-like forms. *d*. Cast-like form, with cells upon surface. *e*. Filamentous forms in a clump.

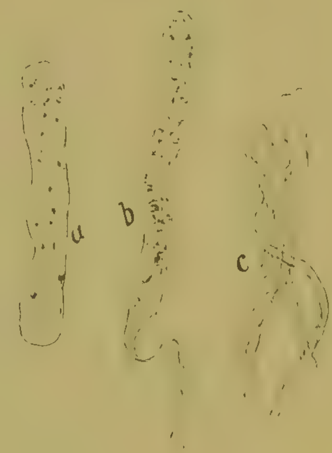
to extend completely across the microscopic field, and if followed out to the end will be found to taper off or gradually become more and more transparent until they cannot be outlined. For this reason too much light should not be used in searching for them, nor should too high a power lens be used. These cylindroids often are grouped in bunches. In other instances we find cylindroids in the form of ribbons, or, in other words, they are wider than the thread-like masses just described. In still other instances the resemblances to true tube-casts are so marked that a differentiation is scarcely possible, except that they are sometimes found to have a filiform tail-like ending.

(See Figs. 160, 161, 162, and 163.) The significance of cylindroids is not definitely known, but they may be taken as an indication of irritation of the kidneys, even if albumin and true casts cannot be found in the urine. They are often seen in the renal irritation following or, rather, accompanying the conditions called lithæmia

FIG. 161.

Non-albuminous urine.
Cast-like forms, with deposit of urates.

FIG. 162.



Non-albuminous urine.
a and b. Cast-like forms. *c*. Filamentous.

or uricæmia, and in that condition in which we find oxaluria. (For further information concerning cylindroids, see Stengel's paper in *The Medical News* of July 15, 1893.)

FIG. 163.

Filamentous and ribbon-like cylindroids.

According to Bramwell, the following is the best method of staining and mounting tube-casts and other urinary deposits. He uses picrocarmine :

"1. An ordinary conical urine glass is filled with equal parts of

urine and an aqueous solution of boric acid, and set aside until the deposit settles.

"2. The deposit is then drawn off by means of a pipette, and transferred to an ordinary test-tube, in which a small quantity (half a drachm is quite sufficient) of picrocarmine solution has been previously placed.

"3. The urine and staining-fluids are then thoroughly mixed by inverting the test-tube two or three times, the end being closed, of course, by the thumb.

"4. The test-tube containing the urine and staining-fluid is then set aside to stand for twenty-four hours.

"5. The deposit, which has by that time settled at the bottom of the test-tube, is then drawn off by a fine-mouthed pipette, placed on a slide, covered, and examined under a low power.

"If any tube-casts are present, they are very easily detected by this method.

"When a cast is detected, it should be carefully brought to the centre of the field and examined with a higher power. If amyloid degeneration is suspected, methyl-violet may be used, for in some cases of waxy disease of the kidney the tube-casts give the characteristic rose-pink reaction with methyl-violet. For permanent preparation the deposit is drawn off as in No. 5, above, and transferred to a small tube of Farrant's medium,[1] in which it remains until the organic deposit has settled, when it is again drawn off and transferred to clear Farrant's solution, whence it is mounted in the usual manner. All organic deposits are thus stained and mounted in a perfectly clear medium. Their minute characters can be studied with the highest powers of the microscope."

The most important sedimentary substances for diagnostic purposes, other than casts, are the products of tissue-changes, or are derived from articles of food. These substances are chiefly the acid urate of sodium and potassium and alkaline urate of ammonium and potassium, uric acid, oxalate of lime, the phosphate, carbonate and sulphate of lime, and the so-called triple phosphates (ammonio-magnesic-phosphate).

The discovery in a urinary sediment of fine shapeless granules,

[1] Farrant's solution is made as follows: Dissolve 1 grm. of arsenious acid in 200 c.c. of distilled water. In this dissolve 130 grms. of gum acacia with frequent stirring, and add 100 c.c. of glycerin. Filter the solution through fine Swedish paper upon which has been deposited a thin layer of talc.

which may be crystalline and shaped like a fan, which are generally brown or pinkish in hue, indicates acid sodium urate. Urine containing such deposits is found to be acid on standing, and will form a brick-dust deposit as soon as the urine is cooled. Acid potassium urate and acid calcium urate, which occur in an amorphous form, are mixed with it in smaller quantities.

The urates themselves have no particular importance except that they are often present in excess in fever, wasting diseases, in gastric disorders, and in attacks of gout.

When in a highly acid urine the student finds rhombic or diamond-shaped plates (Plate XIII., Fig. 2), or plates of a similar shape with the lateral angles rounded off, or quadrate crystals or square plates, or plates like double-headed arrows, or rosettes of crystals, or bundles of crystals like bundles of kindling-wood, these forms are uric acid. Any urine will deposit such crystals if it stands for many hours (say ten hours), as its acidity increases, and therefore the discovery of these crystals only possesses significance if they are found in from four to six hours, as this shows an excess of uric acid, which in turn is found in gouty, rheumatic persons or those who eat to excess and take no exercise. Often an excess of uric acid in the urine antedates the development of chronic contracted kidney. Uric acid also appears in excess in cases suffering from fever and acute inflammations. It is also eliminated in excess in leukæmia, splenic enlargement, hepatic cirrhosis, and gastro-intestinal catarrh. The rosette crystals just named are often found in diabetic urine.

Small square, brilliant, octahedral crystals which are perfectly transparent refract light strongly, and look somewhat like the back of a square envelope at times, are those of oxalate of lime. (See Fig. 164.) The significance of oxaluria is quite important, for it is often a concomitant symptom of melancholia, dependent upon improper metabolism producing such symptoms, and so if present separates this class of cases from those of the true disease melancholia and indicates the use of nitrohydrochloric acid. It is found in the urine of persons who have eaten of peas, cabbage, and tomatoes, and in that of persons suffering from spermatorrhœa. If not due to the ingestion of these foods, oxaluria indicates deficient oxidation of nitrogenous tissues.

Creatin in the urine occurs in very brilliant prisms of a rhomboid

form, the end of which is often split into a frayed end. (See Fig. 165, *a*.) It is not present in normal urine.

Fig. 164.

Oxalate of lime crystals.

Creatinin also exists in normal urine in small amounts in prismatic, colorless, brilliant crystals of the shape shown in Fig. 165, *b*.

Fig. 165.

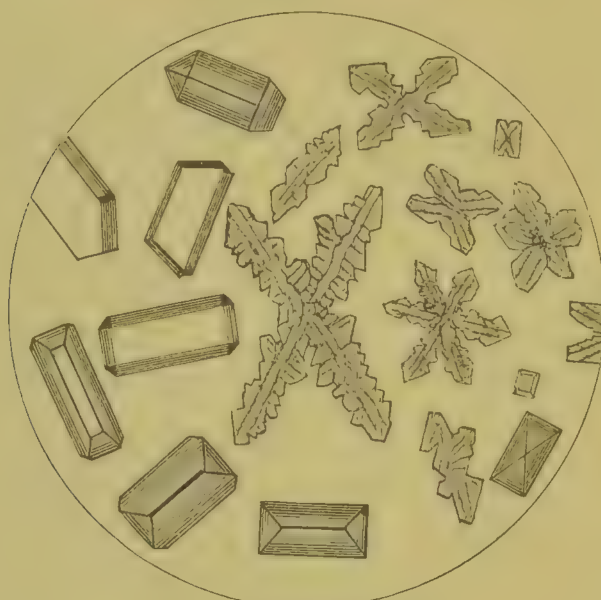
Crystals of creatin and creatinin. (Charles.)
a. Crystals of creatin. *b*. Crystals of creatinin. *c*. Crystals of chloride of zinc and creatinin.

When dark-brown spherical masses covered with thorn-like crystals or sharp spicules are formed in alkaline urine they are composed of ammonium urate (Plate XIII., Fig. 3), and they will be found asso-

ciated with crystals which are flat or shaped like coffin-lids, or more rarely are feathery, star-shaped masses which are large in size. These are the crystals of the triple phosphates. (See Fig. 166.) In addition, such urine will contain amorphous calcic phosphate.

The crystals of the triple phosphates are of some diagnostic importance, as they do not exist naturally in the normal urine, but are formed when the ammonia is set free by the decomposition of the urea. If such crystals are found in freshly passed urine, they indicate that ammoniacal fermentation is taking place in the bladder, a condition often seen in chronic cystitis and in some cases of paraplegia

FIG. 166.



Triple phosphate crystals.

arising from injury to the cord or myelitis. A deposit of the triple phosphates and amorphous calcium phosphate, making a sediment like that of purulent urine, is sometimes seen in overwork of the nervous system and in cases of general debility.

In addition to these amorphous and crystalline bodies found in the urine there are a number of others derived from the body, or due to extraneous contamination. These are epithelial cells derived from the kidneys, ureters, bladder, or urethra (Plate XIII., Fig. 4). Eggs or bodies of several parasites, tubercle bacilli, gonococci and streptococci, or staphylococci, are also sometimes seen under the microscope. In addition, we find spermatozoa in certain cases. (See Fig. 169.)

Thus we may find the embryos of filaria, echinococcus hooklets (Fig. 167), and the eggs of distoma hæmatobium, which are very rarely seen.

Fig. 167.

Echinococcus, with two hooklets, and section of cystic membrane, greatly magnified. (Peyer.)

Tubercle bacilli are to be found by the same staining-processes as when they are sought for in the sputum (see chapter on Cough and Expectoration), and, if found in the urine, indicate renal or vesical tuberculous infection, provided that the patient has not contaminated the vessel holding the urine by sputum infected with the organism. They are not to be confused with the bacilli found in preputial smegma, which look like tubercle bacilli and take the same stains.

Fig. 168.

Streptococci. (Abbott.)

Gonococci indicate the presence of a specific urethritis or vaginitis, and are found by staining and using a $\frac{1}{12}$ homogeneous immersion lens with a No. 2 eye-piece. The process of staining is the eosin and methylene-blue stain. The material on the cover-glass is stained for a few seconds in an alcoholic solution of eosin, then the excess of stain is washed off and the slide is then stained for ten minutes in an aqueous solution of methylene-blue. Streptococci appear in chains and are stained by the same process. They show infection from pus or are found in cases of erysipelas. (See Fig. 168.) Staphylococci also represent pus in the urinary tract.

The presence of spermatozoa is more rare than is generally thought. They may be in the urine either as the result of a true spermatorrhœa, which is rare, or from some of the semen remaining in the urethra after an ejaculation in coitus, or from an emission at night without intercourse. They appear as small transparent bodies having a head and tail and, if alive, possessing very active movements. (Fig. 169.)

FIG. 169.

Spermatozoa, with casts of seminal tubules and spermine crystals.

Fermentation resulting from the presence of a number of special fungi takes place both in healthy and diseased urine after it is passed. In normal urine the acidity, which is generally present to a slight degree, becomes still more acid through the growth of a special fungus. This process is accompanied by the deposition of uric acid, acid sodium urate and calcium oxalate, and also amorphous urates. After the urine is exposed still longer it undergoes an alkaline fermentation, and there develop in the fluid the micrococcus ureæ and bacterium ureæ. As a result the urea takes up water and decomposes with the development of CO_2 and ammonia. No sooner is a positive alkaline reaction established than those ingredients of the urine which are insoluble in an alkaline solution are precipitated, namely, amorphous calcic phosphate, ammonium urate, and ammonio-magnesic phosphate. The first is amorphous, but the ammonium urate appears under the microscope in the form of small granules of a dark color

which are covered with spines. The crystals of ammonio-magnesic phosphate are shaped like a coffin-lid, and are large.

The third form of fermentation taking place in the urine is that which occurs in diabetic urine, and is due to the saccharomyces albicans, the micro-organism which produces fermentation in ordinary solutions of glucose.

Chemical Tests. The chemical tests of the urine give us much important information. We commonly test it for albumin and for sugar, and, if we wish still further information, we examine it for its percentage of urea, uric acid, and for its peptones, or, to use a better term, its albumoses.

ALBUMINURIA. There are a great number of tests for albumin and sugar. Many of them are open to fallacies, and they are therefore to be avoided by the busy practitioner, who can rest assured that if he finds no albumin by the heat and nitric-acid test, if properly carried out, that he can put albuminuria out of the possibilities of the case, provided he tests samples taken at different times and on several different days, for sometimes albuminuria is intermittent.

The best test consists in taking filtered urine and pouring enough of it into a perfectly clear test-tube to fill it about two-thirds. To this are now added a few drops of acetic acid to render it acid; for, if neutral, the albumin will not be coagulated by heat. The upper part of this urine is now boiled by holding it over an alcohol lamp, and if albumin is present a fine cloud will appear in the boiled part of the urine, while the lower part remains clear. This cloud may be due to albumin or earthy phosphates. If a drop or two of nitric acid is allowed to trickle down the side of the tube, the cloud is dissipated if due to phosphates, but not changed if due to albumin.

If the urine be turbid before the test by reason of an excess of urates, the fluid can be rendered clear by gently heating all of it.

Heller's test of adding a few drops of urine to HNO_3 in a test-tube is too fallacious to be used.

The quantitative tests for albumin are many of them impractical for the busy doctor. The best method is by means of percentage-tubes placed in a centrifuge-machine. By this means all the albumin is thrown down. The tubes are filled to the 10 c.c. mark with urine and $2\frac{1}{2}$ c.c. of potassium ferrocyanide solution (one part to ten) are added. Next we add $1\frac{1}{2}$ c.c. acetic acid and thoroughly mix all these liquids, and the tube being placed in the centrifuge the machine is worked till all the albumin has settled. Each $\frac{1}{10}$ c.c. mark on

the tube represents 1 per cent. by bulk of albumin; that is, if the albumin extends up to the $3\frac{1}{2}$ cubic centimetre mark the albumin amounts to 35 per cent.

The significance of albumin is not as grave in all cases as it was considered at one time, nor is its quantity of great import necessarily, for in some of the gravest cases of renal disease, as chronic contracted kidney, it is excreted in very small amount, and it occurs in the urine sometimes in large quantities without any kidney lesion being present. As a rule, however, it indicates renal disease in one of its inflammatory forms, provided it is associated with other renal symptoms. It may depend on changes in the blood in which the diffusibility of its albumin is increased (Semmola), and we see albuminuria in cases of anæmia and in convalescence from long disease or from the effects of poisons. Again, circulatory changes may cause albuminuria by causing congestion of the kidney, as in cases of failing heart from its various causes. There is an intermittent, little understood, form of albuminuria, called cyclic albuminuria, or the albuminuria of adolescence, in which the albuminuria is absent on rising from bed in the morning, but appears if exercise is taken. An excess of albumin in the diet may cause albuminuria.

Sugar in the Urine. The presence of sugar is determined by a large number of tests, of which the simplest and most reliable are Haines's test and the test of Whitney. Haines's test consists in making a solution as follows: Pure copper sulphate, thirty grains; distilled water, half an ounce; and thoroughly dissolve the copper in this water; then add pure glycerin, one-half an ounce, which is to be thoroughly mixed; and then add liquor potassa, five ounces. One drachm of this is to be placed in a test-tube and gently boiled, and to this are now added six to eight drops of the urine, and the liquid again gently boiled. If sugar is present, a copious yellow precipitate is formed. This is better than Fehling's test, because it is a permanent fluid.

Whitney's test is a solution of ammonio-cupric sulphate, of which one drachm is decolorized by $\frac{1}{30}$ grain of glucose. The solution to the amount of one drachm is placed in a test-tube and heated to the boiling-point. The urine is now added drop by drop. If no sugar is present, no change will occur; but if it is, the blue color will begin to fade, and finally the liquid will become perfectly colorless. As the fading process begins the urine should be added more slowly, three to five seconds of boiling intervening between each drop. If

there is any shade of blue or green left in the solution, reduction has not taken place. The following table shows how this test may be used for the quantitative estimation of sugar:

If reduced by	*It contains to the ounce.*	*Percentage.*
1 drop	16 or more grains.	3.33
2 drops	8	1.67
3 "	5.33	1.11
4 "	4	0.83
5 "	3.20	0.67
6 "	2.67	0.56
7 "	2.29	0.48
8 "	2	0.32
9 "	1.78	0.37
10 "	1.60	0.33

If the urine contains more than 3.33 per cent. of sugar, it is to be diluted by from one to ten parts of water, and the amount found in the table multiplied by the amount of dilution.[1]

As Fehling's test is so widely used it must be described. Wickham Legge thus describes it:

This solution may be prepared in the following way: 665½ grains of crystallized potassio-tartrate of sodium are dissolved in five fluid-ounces of a solution of caustic potash, sp. gr. 1.120. Into this alkaline solution is poured a fluid prepared by dissolving 133½ grains of sulphate of copper in ten fluidrachms of water. The solution is exceedingly apt to decompose, and must always be kept in stoppered bottles and in a cool place. It is usually, therefore, more convenient not to mix the alkali and copper until the solution be wanted for use. In this case, a fluidrachm of the sulphate of copper solution may be added to half a fluid-ounce of the alkaline solution prepared as above.

About a couple of drachms of this test-solution are poured into an ordinary test-tube and the fluid boiled over a lamp, and set aside for twelve hours. If no deposit forms, the solution may be used for analysis; but if a red precipitate be thrown down, the liquid has decomposed, and a fresh supply must be had.

While the solution is boiling in the test-tube the urine must be added to it, drop by drop, and the effect watched. A few drops of a sample of urine which contains a large percentage of sugar will at once give a precipitate of yellow or red suboxide of copper; but if no precipitate occur, the urine should be added to the fluid, drop by drop, any deposit being carefully looked for, until a quantity equal

[1] This test, under the name of Aquæ Sapphirina, can be had of the Lewis Chemical Co., of New York.

to that of the Fehling's solution employed has been added. If no precipitate be found after setting the test-tube aside for an hour, the urine may be considered free from sugar.

Cautions: 1. The test-solution should never be used without boiling beforehand for a few seconds; the tartrate being exceedingly apt to decompose, and the solution then reduces copper as effectually as would grape-sugar.

2. The quantity of urine used in the test should never be greater than the quantity of test-solution employed.

3. After adding urine in volume equal to the Fehling's solution, the boiling of the mixture must not be continued, as other bodies present in the urine, besides sugar, will reduce copper at a high temperature.

If the examination for sugar is to be made with the greatest care, the urine should always be filtered, at least three times, through animal charcoal. This removes all urates and uric acid, which often partly reduce the Fehling's solution, but the sugar goes through the filter. Sir William Roberts directs that the Fehling's solution be placed in a test-tube to the depth of about one-quarter inch and the filtered urine added to the depth of two inches, and the two fluids well mixed. The flame of the lamp is then applied to the upper part of the urine, as in testing for albumin, and this is briskly boiled for a few seconds. The test-tube is now held up to the light, and, if sugar is present, the upper part has a yellowish tinge, while the earthy phosphates are thrown down in golden-colored flocculi.

The quantitative estimation of sugar is best made by the Whitney test, already described, or by the fermentation-method of Roberts, which depends upon the principle that grape-sugar is decomposed into alcohol, carbon dioxide, etc., by the fermentation set up by yeast. As a result of this the urine loses its specific gravity, and each degree of specific gravity has been found to equal one grain of sugar in the fluidounce. In other words, if the specific gravity before the test was 1.035 and after the test 1.015, the amount of sugar present would be twenty grains per ounce. Four ounces of urine are placed in a twelve-ounce bottle and a lump of German yeast added. The bottle is then corked with a perforated cork to permit the gas to escape, and placed in a warm place for twenty-four hours. By its side is placed a tightly corked bottle of the same size, holding four ounces of urine and no yeast. The specific gravity of both specimens is taken simultaneously, and the difference in degrees represents the

number of grains of sugar in each ounce. The loss in degrees of specific gravity multiplied by 0.23 will give the percentage of sugar.

The significance of sugar in the urine is various. If it is persistent and accompanied by wasting, polydipsia, and polyphagia, it is a sign of diabetes mellitus, due to a lesion in the medulla, to morbid functional activity of the liver, or to changes in the pancreas. If in a young person, the prognosis as to life is fatal; if in middle age, it is hopeful; if in persons after fifty, it is quite favorable.

Sugar is sometimes found in small amounts in the urine of very obese persons, and its presence, under these circumstances, does not necessarily indicate a grave prognosis; but, on the other hand, there are cases of so-called diabetogenous obesity in which the prognosis is very grave. They are to be separated from the class just named by the fact that the systemic symptoms of wasting, depraved nutrition, itching, furunculosis, and profuse diuresis are present. Then, too, in the latter form, the disease is usually associated with obesity in early life, whereas in the milder form it occurs in the obesity of advanced life. Diabetes occurring in old age, or after sixty years of age, has not the grave prognosis attached to it that exists in connection with the disease in earlier life. The younger the patient the graver the malady.

The other indications of glycosuria are of little importance. Glycosuria occurs in many infectious diseases and after the ingestion of some poisons, notably phloridzin.

Albumose in the Urine. Albumoses, or peptones, in the urine may be tested for and their presence recognized by saturating slightly acidified urine with ammonium sulphate, filtering out all precipitate, and adding to the filtrate very gently a little solution of picric acid, seven grains to the ounce of water. Any precipitate is peptone.

A better method than this, however, is that described by Harris, in which one part of albumose in 5000 parts of urine can be recognized. Before the test is made every trace of coagulable albuminoid matter must be removed from the urine to be tested. This is done, to use Harris's words, as follows:

To 20 c.cm. of (acid) urine[1] in a test-tube are added six or eight drops of a saturated solution of salicyl-sulphonic acid in distilled

[1] The urine must be fresh. If it must stand several hours before it can be examined, it should be preserved from the growth of bacteria in it by the addition of some antiseptic, preferably a few drops of formalin, which will keep it several days, and does not interfere with subsequent tests.

water, and 1 grm. of chloride of lead. Shake well and boil about thirty seconds. Cool by shaking in running water from the cold-water tap.

Filter through ordinary clean, white filter-paper until the urine is clear. Now add a few drops of a clear, saturated solution of sodium sulphate in distilled water, in order to precipitate what lead is held in solution ; raise to the boiling-point, and cool under the cold-water tap as before.

Filter again until clear. We should now have a perfectly clear urine, absolutely free from every trace of coagulable albuminoids, including nucleo-albumin, in which we may search for albumose or peptone. This clear filtrate is divided into three equal portions and placed in test-tubes, one of which is kept for comparison, the other two for further analysis.

To one of these are now added three or four drops of a saturated solution of salicyl-sulpho-tungstate of sodium in distilled water.[1]

If albumose or peptone be present, a cloudiness will appear, varying in degree according to the amount of these proteids present.

As the amount of albumose present is often very minute, it may be necessary to compare the tube with the control-tube in order to detect the cloudiness.

The cloudiness disappears entirely on gently heating the test-tube, to reappear on cooling.

To the third tube the test is varied by allowing about 5 c.cm. of a dilute solution of the salicyl-sulpho-tungstate of sodium (made by adding about ten drops of the strong solution to 5 c.cm. of distilled water) to flow very gently down the side of the tube, so as to rest on the urine as a separate layer.

This should be very carefully done so the line of contact will be sharp and clear-cut, not diffuse. A cloudy line appears at the point of contact of the two liquids if albumose or peptone be present.

When the amount present is very small it may take two or three minutes for the line to develop, and shows best, the two liquids being clear, when held in front of a dark background.

As before stated, this test is extremely delicate, 1 part in 50,000

[1] Salicyl-sulpho-tungstate of sodium is prepared as follows:
To a boiling saturated solution of tungstate of sodium in distilled water salicyl-sulphonic acid is gradually added, under constant stirring, until the solution no longer turns red litmus blue; or, in other words, until the alkaline tungstate of sodium is completely neutralized. Upon cooling the salicyl-sulpho-tungstate of sodium crystallizes. A solution is now made of this in cold, distilled water and filtered. A perfectly clear, colorless fluid results.

being readily detected; it is simple, and can be easily applied in fifteen to twenty minutes.

Owing to the delicacy of the reactions it is necessary that all test-tubes be absolutely clean and the test-solutions perfectly clear, otherwise a slight reaction may be easily overlooked.

The sodium sulphate solution must be added in slight excess in order to insure the precipitation of all lead, as any lead left in solution would be precipitated by the salicyl-sulpho-tungstate of sodium, and thus interfere with the test.

This would be easily recognized, as the cloudiness in that case would not disappear on heating, but become more marked.

The boiling during the application of the test, while not absolutely necessary, very materially facilitates the reactions and should always be done. The cooling, after boiling and before filtering, must never be omitted.

If these few simple points be carefully observed, no difficulties will be experienced in applying the test.

The significance of albumosuria is various. It is present in croupous pneumonia, all suppurative processes, empyema, tuberculosis, small-pox, mumps, erysipelas, cancer of the viscera, jaundice, and apoplexy, and in typhoid fever and phosphorus-poisoning. Von Jaksch asserts that it is present in epidemic cerebro-spinal meningitis and absent in tubercular meningitis, and that it is a positive differential sign of the former disease if no ulceration of the lung is present. Harris, on the other hand, asserts that albumosuria is simply a manifestation of the action of micro-organisms, and is thus only an indication of an infective process.

It ought not to be forgotten that albumosuria occurs in the normal puerperium.

The Urea in the Urine. The amount of urea is to be estimated by the process of Lyons, as follows (Fig. 170):[1]

1. A bottle is provided with perforated rubber cork and delivery tube; in this the decomposition of the urea is effected.

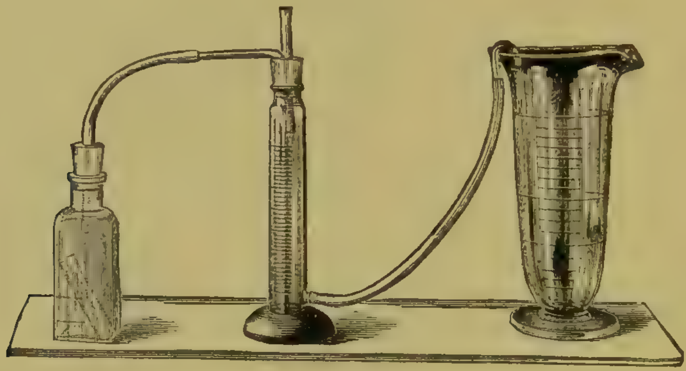
2. A small test-tube to contain the urine, graduated to hold 4 c.c., the quantity employed in each experiment.

3. A graduated jar for measuring the gas evolved. The jar is provided at the bottom with an "overflow" tube, and at the top

[1] This apparatus, with full directions for use, can be obtained from Parke, Davis & Company, Detroit, Mich., for one dollar.

with a vent-tube closed with a rubber cap, to secure accurate adjustment of the level of the fluid in the jar at the commencement of the experiment.

FIG. 170.



Ureometer.

The process is as follows: Put into the square bottle 20 c.c. of a special solution of chlorinated soda (for formula see below), and add 5 c.c. of a 20 per cent. solution of potassium bromide; fill the test-tube exactly to the mark (4 c.c.) with the urine to be examined, and lower it into the bottle by means of a thread or by the aid of a pair of dressing-forceps, taking care that none of its contents are spilled in the operation. Fill the graduated jar with water, which must be of the same temperature as the air of the room, to a point a little above the 0° of the scale, supporting the extremity of the overflow-tube so that no water can escape. Remove the rubber cap from the vent-tube and connect the apparatus, pressing in the rubber corks firmly so as to make the joints air-tight. Finally, put on the rubber cap, drawing it down so as to force a little water out of the overflow tube, and bring the level of the water remaining exactly to the 0° mark, the orifice of the overflow-tube being on the same level. A little practice will make this easy.

To make sure that the connections are all perfectly air-tight, lower the end of the overflow-tube a few inches; a few drops of water will escape from diminished pressure; but if the joints are perfect, there will be no further dropping. If there is any leakage, the defective joint must be found, and the difficulty corrected before proceeding further with the experiment. Having made sure that the connections are perfect, catch the curved end of the overflow-tube over the edge of a measuring-graduate, as shown in the illustration (an ordinary bottle or any other receiver may be used in place of the graduate). Now, by canting the bottle, cause the urine to

flow out of the test-tube and mix with the test-solution. Effervescence is at once produced, and the gas evolved forces a corresponding volume of water out of the overflow-tube. Shake the bottle occasionally to promote the escape of the gas. When the action appears to be at an end pour into the measuring-graduate enough water to reach above the opening of the overflow-tube, in order that cooling of the gas evolved, which is at first quite warm, may not draw air into the apparatus. Let the apparatus stand fifteen or twenty minutes to cool, then shake the bottle containing the urine once more and proceed to read off the result. To do this, it is necessary to bring the opening at the end of the overflow-tube just to the same level as that of the fluid remaining in the graduated cylinder, since raising or lowering the tube slightly affects the volume of the gas to be measured. The percentage of urea is read off without need of any calculation from the scale of the instrument. The accompanying table will enable the physician to ascertain from the percentage-amount of urea in the specimen examined what is the absolute amount of that compound excreted during the day, provided, of course, the whole of the urine passed during the twenty-four hours has been collected together and carefully measured.

Per cent. of urea by ureometer.	Quantity of urea in grains in 1 fluidounce.	Per cent. of urea by ureometer.	Quantity of urea in grains in 1 fluidounce.
0.1	0.456	1.9	8.658
0.2	0.911	2.0	9.114
0.3	1.367	2.1	9.570
0.4	1.823	2.2	10.025
0.5	2.279	2.3	10.481
0.6	2.734	2.4	10.937
0.7	3.190	2.5	11.393
0.8	3.646	2.6	11.848
0.9	4.101	2.7	12.304
1.0	4.557	2.8	12.760
1 1	5.013	2.9	13.215
1.2	5.468	3.0	13.671
1.3	5.924	3.1	14.127
1.4	6 380	3.2	14.582
1.5	6.836	3.3	15.038
1.6	7.291	3.4	15.494
1.7	7.747	3.5	15.950
1.8	8.203		

EXAMPLE.—The patient has passed 24 fluidounces of urine, found to contain 2.4 per cent. of urea. The total urea excreted will therefore be 10.937 (from the table) × 24 = 262.488 grains.

For exact estimations the temperature of the room in which the experiment is made must be about 70° F. (21° C.). A variation from this temperature of 20° will, however, make a difference in

the result of only about 0.2 per cent., so that the temperature-correction may be regarded as unimportant.

In the process given for the manufacture of the test-solution the hypochlorite is changed into hypobromite.

This mixture gives more uniform and trustworthy results than those obtained with the chlorinated soda alone, which is recommended by Dr. Squibb. It is in fact identical in its action with the hypobromite solution, without the great inconvenience of handling bromine. A few minutes must be allowed to elapse after the mixture is made before mixing the urine with it; but this need occasion no delay, since the mixture can be put into the bottle before filling the cylinder and making the connections.

The activity of the solution of chlorinated soda can be easily tested by adding to a little of it in a test-tube a few drops of the solution of potassium bromide, and then a little muriate of ammonium, which should cause brisk effervescence. If this is not the case, it is too much deteriorated for use.

In some rare instances it will happen that the urine contains a larger proportion of urea than the ureometer is capable of indicating. When this is the case, and in general when the specific gravity of the urine exceeds 1.030, sugar being absent, it will be best to dilute the urine with an equal volume of water before making the test. Four c.c. of the diluted urine will then be used as usual in the experiment, but the percentage given by the reading of the instrument must be multiplied by two.

It will be found in practice that an estimation of urea by this apparatus consumes very little time, and the results for all practical purposes are as accurate as could be wished for.

Formula for Special Solution of Chlorinated Soda. Shake chlorinated lime (best quality) 12 grammes with water 100 c.c.; let settle and filter into a 250 c.c. bottle. Wash the residue with enough water to obtain 130 c.c. of clear filtrate.

Dissolve sodium carbonate 24 grammes in water 45 c.c. Add this solution to the above filtrate, mix thoroughly, and, when reaction is complete, filter, passing, if necessary, enough water through residue on filter to obtain 165 c.c. of filtrate.

The clinical value of estimating the urea is great in cases of renal disease, and it is also of value in diabetes mellitus and during pregnancy or in the puerperium and before surgical operations. The quantity of urea excreted in twenty-four hours is increased in nearly

all fevers and inflammations, and is decreased in any cachectic state in which the metabolic changes in the tissues are impaired. It is also decreased in diseases which greatly modify the activity of the liver, the gland which makes urea. The particular value of estimating the urea in Bright's disease and in pregnancy lies in this, namely, that the renal disorder in these conditions results in an imperfect elimination of urea, and as a result it, or closely allied products, are retained in the blood. If, therefore, in a pregnant woman or a person suffering from Bright's disease, analysis shows a constant diminution in the amount of excreted urea, the physician is warned that a uræmic convulsion or other manifestation of uræmic disorder is imminent, and can take active measures to relieve the patient, for, after the uræmia is developed, treatment is of comparatively little value.

Although the quantity of urea varies very greatly in perfect health, the mean amount excreted in twenty-four hours by a healthy man of twenty to forty years is about 512 grains. Women excrete a little less than men and children still less in actual quantity, but more in proportion to their weight.

It is absolutely necessary in estimating the amount of urea excreted in twenty-four hours to test a sample of the urine obtained from all the quantity passed in that time, as a test of the urine passed on one occasion is no guide for the total daily quantity.

Chlorides in the Urine. The urine in health contains chlorides of sodium and potassium, and these are to be discovered by placing a fluidrachm of urine in a test-tube and then adding a drop of nitric acid, and finally a few drops of a solution of nitrate of silver. If chlorides are present in considerable quantity, a white precipitate of chloride of silver is thrown down, which can easily be distinguished from albumin; but if some doubt is felt as to its character, the addition of a little caustic ammonium will redissolve it if composed of chlorides, and it will be reprecipitated if nitric acid is again added. If the same quantities of urine and reagents are taken daily and placed in a test-tube of equal dimensions and the precipitate allowed to settle for twenty-four hours, we can gain an approximate estimate of the relative quantity of the chlorides. The amount ordinarily passed in twenty-four hours by a healthy man is 250 grains.

The clinical significance of a decrease in the chlorides is not great. They are decreased in the acute stages of croupous pneumonia, acute

articular rheumatism, and some other fevers; and if they gradually increase, they indicate the development of convalescence.

THE TOTAL QUANTITY OF SOLIDS excreted by the kidneys in twenty-four hours can be roughly estimated by what is known as Haines's modification of Haeser's method. If the solids are much decreased, more accurate methods of testing should be resorted to. The method just referred to is carried out as follows: The last two figures of the specific gravity of the urine are multiplied by the number of ounces voided in twenty-four hours, and the product is multiplied by one and one-tenth. Thus, if a patient passes 32 ounces, and the specific gravity of the urine is 1.012, we multiply 32 by 12, which equals 384, and this is multiplied by 1.1, which equals 422, which would be much less than the normal for a person of, say, 150 pounds, who should pass about 1150 grains of solids in twenty-four hours.

Ehrlich has claimed that a distinct aid to the diagnosis of enteric fever can be obtained by the so-called diazo-reaction of the urine, although it is to be remembered that this takes place in several other conditions of the body, notably pulmonary tuberculosis, measles, pyæmia, scarlet fever, and erysipelas. It is usually present only in severe cases of these ailments. Further than this, Ehrlich asserts that the reaction is usually to be obtained about the fourth to seventh day of the disease. A faint reaction is indicative of a mild attack.

The test is as follows:

1. Take 2 grammes (30 grains) of sulphanilic acid, 50 c.c. of hydrochloric acid, and 1000 c.c. of distilled water. 2. Take a solution of sodium nitrite in water of the strength of 0.5 per cent. Fifty parts of No. 1 and one part of No. 2 solution are now placed in a test-tube and an equal amount of urine added, and this mixture is then rendered strongly alkaline by strong ammonia-water. If the diazo-reaction is present, the liquid becomes carmine-red in color; and if the test-tube is shaken, this color is seen in the foam. This, the coloration of the foam, is the point in the reaction, for, if the liquid only is red, the test is not positive. After standing a day a green precipitate will form in the tube.

The Manifestations of Urinary Disorders.

Having considered the pathological changes found in the urine and their significance, we now pass on to a consideration of the general

symptoms which will usually be found associated with these variations from the normal functional activity of the urinary organs.

Let us suppose that a patient presents himself complaining that he has been seized with pain in the small of the back, and perhaps by nausea and chilly sensations, followed by a marked decrease in the quantity of urine secreted, which decrease may actually amount to suppression of the urine. The urine that is passed is high-colored or smoky in hue, sometimes looks like porter, and forms a very heavy sediment on standing. If it is filtered and tested for albumin, it will be found to contain this abnormal ingredient in large amount, and a microscopical examination of the sediment will reveal a large number of blood-corpuscles, epithelial cells, and casts (hyaline) made up of blood-cells, epithelium, and albumin. Scarcely will these signs have been noted when the patient will be seen to be anæmic, and puffiness of the face about the eyes will be evident. This puffiness may then pass on to a general anasarca, but it is to be remembered that the most violent *acute diffuse nephritis* may exist without developing anasarca. If the disease be in a child and it is due to scarlet fever, anasarca is common, as is also uræmia. The pulse in patients with this form of nephritis is usually hard and tense, and the sharp and clear second sound of the heart, as heard at the second right costal cartilage, will indicate the high arterial tension. The skin is generally dry, and, it may be, harsh to the touch. Should the symptoms persist for over a month the possibility of the disease becoming chronic renders the prognosis doubtful; but as a rule, particularly in young persons, the prognosis of acute diffuse nephritis is favorable. In the acute diffuse nephritis of pregnancy the prognosis is, of course, grave. The history of the case prior to the attack of this ailment will usually be that the patient has been exposed to cold or wet, has been or is a sufferer from an acute infectious disease, has swallowed or inhaled some irritant poison, or has suffered from some severe burn of the surface of the body.

If, instead of an acute attack of illness, the symptoms just described come on gradually and insidiously and the tendency to anasarca is marked and persistent, we have before us a case of *chronic parenchymatous nephritis,* in which the prognosis is most grave. Uræmic vomiting and coma may occur in this class of patients (see chapter on Vomiting). Blood-cells are also found in the sediment of the urine in these cases, but are not so numerous as in acute diffuse nephritis.

A set of symptoms which differ very markedly from those just described occur in cases of *chronic contracted kidney* (chronic interstitial nephritis). The following description of the symptoms may be taken as representing a typical case: The patient, who is usually past middle life, finds that he or she urinates more frequently and passes a greater amount of urine than heretofore. Often the sleep is disturbed by the necessity of arising to urinate. Instead of the urine being heavy and clouded, it is unusually clear and limpid; and in place of the high specific gravity of diffuse parenchymatous nephritis, we find it unusually low (only 1.010 to 1.015). Albumin is only found inconstantly and in traces, and is generally to be sought for in the urine passed by the patient when first arising from bed. The pulse is usually much increased in tension, and atheroma of the bloodvessels is more or less marked. This high-tension pulse is a valuable diagnostic sign. The heart, which in acute diffuse nephritis may be slightly dilated, or in chronic parenchymatous nephritis somewhat hypertrophied, is in this disease usually markedly hypertrophied, and the second sound at the second right costal cartilage is accentuated. In addition to these symptoms we find that chronic bronchitis is not rare, and that pulmonary œdema and attacks of shortness of breath are often present, the latter being most marked at night. Uræmic symptoms are more commonly seen in this class of cases than in any other, and violent vomiting difficult of control should always make the physician test the urine to discern renal mischief. Unlike parenchymatous nephritis, dropsy is a rare complication of chronic contracted kidney. Microscopic examination of the urine will only reveal a few hyaline and granular casts.

The prognosis as to cure is bad, but life may be prolonged indefinitely.

A copious flow of urine of a low specific gravity and of a pale, clear appearance, containing fatty, hyaline, and finely granular casts, is often seen in cases of *amyloid disease* of the kidney, and the presence of syphilis, of prolonged suppuration, or extensive bone disease, due, it may be, to tuberculosis, with concomitant enlargement of the liver and spleen, separates it from any other ailment. Albuminuria may be a marked or an absent symptom.

Let us suppose, however, that a patient comes to us with a history of exceedingly copious urination, of great thirst, of loss of flesh, and has a dry, harsh skin, we immediately recognize that a test

of the urine will probably reveal the case to be one of *diabetes mellitus.* This will be pointed to if a high specific gravity is found present in a clear limpid urine, and confirmed if the tests for sugar already given produce a reaction. If the urine has a constant low specific gravity and contains no albumin or sugar, the case is probably one of diabetes insipidus. The other prominent symptoms of diabetes mellitus are furunculosis, intense itching and erythema (see chapter on the Skin), an excessive appetite, and, in severe cases, gangrene of the extremities or diabetic coma (see chapter on Coma and Unconsciousness).

Should much pus be present in the urine it is probably derived from a *pyelitis* or a suppurative inflammation of the pelvis of the kidney. The symptoms of this state are, briefly, a constant or intermittent pyuria, usually an acid reaction of the urine, chills and fever, which may mislead the physician into a diagnosis of malarial poisoning, or, in other cases, if the pyelitis be tubercular, hectic fever may be present. Sometimes violent attacks of pain resembling renal colic are passing symptoms, and not uncommonly an anæmia and loss of strength are notable. There is often pain in the back, which is made worse by pressure with the hand, and, rarely, if the suppurative process be marked, typhoid symptoms may be present.

If the pyelitis be tubercular, tubercle bacilli may be found in the urine. If due to a calculus, there may be a history of gravel and renal colic. Pyelitis is to be separated from cystitis by the fact that in it the urine is acid, in cystitis it is ammonical; by the pain in the renal region, often unilateral; and by the use of the cystoscope. The prognosis varies. If due to an infectious fever, recovery usually occurs. Tuberculous pyelitis may also recover.

CHAPTER XIII.

THE BOWELS AND FECES.

Constipation and diarrhœa—The causes of these two symptoms and their diagnosis—The diseases in which these symptoms occur—Choleraic diarrhœas—Dysentery—The color of the feces—Intestinal parasites.

THE consideration of the condition of the bowels and feces as indicative of disease affecting the intestines themselves and other organs closely associated with their functions, can be best divided into several parts, namely, the functional disorders of the intestines and the organic diseases from which they may suffer, on the one hand, and the appearance of the feces in both functional and organic diseases of the abdominal viscera in general, on the other. The most common forms of intestinal disturbance are constipation and diarrhœa.

CONSTIPATION may be due to mere sluggishness of bowel-movement because of both nervous and muscular atony, or to deficient secretion of the intestinal juices, or, again, to the too rapid absorption of the liquids from the fecal matter while it is passing through the colon. It is also associated with all those conditions which prevent the proper secretion of bile, which liquid very materially increases peristalsis. Thus, we see obstinate constipation in most cases of jaundice, catarrhal or obstructive; in cases of hepatic disease, producing a deficient biliary flow; and in phosphorus-poisoning, in which the fatty degeneration and hepatitis prevent biliary secretion. Further than this, the constant ingestion of foods which are absorbed nearly *in toto*, or, in other words, leave little residue, particularly raw or boiled milk, produces constipation. Again, the use of wines containing large amounts of tannic acid may produce similar results because of the astringency of this substance, and chronic constipation from the use of large quantities of badly infused or boiled tea made with hard water is frequently met with. When too rapid absorption of the liquids takes place from the feces the cause may be lack of liquid ingested, and the remedy be full draughts of pure water; or, again, constipation occurs as a manifestation of diabetes insipidus or diabetes mellitus, because the polyuria of these affections drains the body of

liquid. Obstinate constipation should therefore always call the physician's attention to these affections and to two other possibilities, namely, that the condition depends upon wilful disregard by the patient of the calls of nature, so that the bowel is forced to retain fecal matter until it becomes hard and dry; or, quite as important, that the constipation may be due to some reflex cause, which, as the result of irritation, results in an arrest of peristaltic movement. Thus, a woman with ovarian and other pelvic trouble may have obstinate constipation which yields little, if at all, to purgatives, but readily to nervous sedatives or even to an opiate. Or, again, in chronic lead-poisoning the inhibitory fibres of the splanchnic nerves may be so irritated that peristalsis is impossible. Here a hypodermic injection of morphine may make a movement possible.

The organic diseases of the bowel producing constipation are many and of great importance. They consist in intestinal obstruction in all its forms, as by bands, by growths, by the process of intussusception, by volvulus, by cicatricial contractions, and by impacted foreign bodies or fecal matter. The presence of a sudden attack of constipation, or the presence of this condition in a degree which fails to yield to mild laxatives, should always put the physician on his guard lest some such grave condition is present. As severe and, finally, stercoraceous vomiting is a fairly constant and more marked symptom of intestinal obstruction than is constipation, a discussion of the various symptoms of intestinal obstruction will be found under the chapter on Vomiting, and the diagnosis of growths of the intestine will be found in the chapter on the Abdomen.

Aside from these causes, it is manifestly impossible to discuss all the conditions of the system in which constipation may be present. The physician must always bear in mind that constipation often results in the absorption of all sorts of poisonous materials from the bowels, which in turn may produce all sorts of symptoms, nervous or otherwise, from epileptiform attacks, in rare cases, to severe headache and vertigo, with vomiting, in others.

Diarrhœa of an acute type depends, as a rule, upon one of four causes, namely, the presence of irritant material in the bowel, which the intestines attempt to get rid of by increased secretion and excessive peristalsis; to relaxation of the bloodvessels of the intestine, with profuse serous leakage and consequent watery purging; to acute inflammation, with excessive secretion of mucus; and to the endeavor of the system to eliminate poisons in this manner, as in

cases of sudden profuse diarrhœa, in cases of chronic renal disease, in which the purging is an effort at elimination.

Such forms of diarrhœa as these are usually sudden in onset and speedily get well of themselves, and it is a mistake to check them too suddenly.

It is impossible to speak of all the possible causes of diarrhœa, or of all the diseases in which it is met with. Only those in which it is a prominent symptom, or one of importance, can be discussed.

One of these is cholera morbus, a disease which manifests itself in profuse watery purging, accompanied by violent pain in the belly, and, after several stools have passed, in a considerable amount of tenesmus. Mucus is almost entirely absent from the dejecta, but particles of undigested food may be found in them. Vomiting is often a severe and simultaneous manifestation of the gastro-intestinal disorder which results in these symptoms, and, if the attack be very severe, it is practically impossible to separate it from true cholera Asiatica if an epidemic of that disease is present. The patient speedily becomes cold and pinched-looking, exceedingly weak, and finally passes into collapse. The pulse becomes feeble, rapid, and running; the face livid, and finally the patient may develop the *facies Hippocratica.* The urine is greatly decreased or entirely suppressed, because of the watery purging and possibly by reason of the effects of certain poisons upon the kidneys. In the great majority of cases the symptoms are not so severe as this, and complete recovery ensues as soon as the offending materials are passed out of the bowels and the patient has time to convalesce.

When an attack of diarrhœa, such as has just been described, comes on in a young child it is usually called cholera infantum, or summer complaint, and it is nearly always due to improper feeding or to the unintentional use of bad food or bad milk. The stools of the child are usually at first filled with curds of milk and green masses, looking as if the curds had been stained with grass-juice or spinach. The child often passes with extraordinary rapidity into a state of collapse, and may die in a few hours or days. The tenesmus often becomes constant and is a distressing symptom, and the tissues become shrunken to a marked degree. The child manifests not only the evidences of the results of profuse purgation, but, in addition, is evidently intoxicated by the toxins absorbed from the bowels, so that it lies on the lap of the nurse in a relaxed and torpid state. The surface of its body is often abnormally cold, and its extremities

may be pinched and blue, but the temperature of the internal organs is generally abnormally high, so that while the axillary temperature may be below normal, the thermometer will reveal a temperature of from 102° to 103° in the rectum. Sometimes the head becomes retracted, as if meningitis was present. The respirations may be sighing or of the Cheyne-Stokes type.

If the child or adult is seized with symptoms such as those described under *cholera morbus* or cholera infantum, and a suspicion of the presence of true cholera is raised, are there any facts which will point to the correct decision in a case, even if, as already stated, a positive differential diagnosis cannot be made? In the first place, a train of symptoms of a malignant type point to the true cholera, rather than cholera morbus or cholera nostras, as it is sometimes called. Again, the evidences of infection or general systemic disease indicate the epidemic malady rather than does a profuse diarrhœa alone. Thus the systemic signs of infection may be so great that death from infection occurs before diarrhœa even begins in the true cholera. Again, it would be possible to determine the presence of true cholera if the comma-bacillus could be demonstrated; but this requires the examination of the fecal matter to be made by an expert, who is familiar with the technique of examining fecal matter for the germ and the necessary measures for their artificial culture.

Symptoms identical with the more violent forms of cholera nostras or true cholera may be produced by *acute poisoning by antimony*, except that in this case we often have profuse sweating and salivation early in the attack. The same symptoms of vomiting, purging of rice-water stools, collapse, cramps in the calves of the legs, and violent pain in the abdomen may be present. A differential diagnosis, without the history of the patient having taken poison is impossible, except by a chemical analysis of the vomited matter, the stools, and the urine, which will contain antimony. The utmost care should be used that the vessels which receive these materials are chemically clean, that they are hermetically sealed until ready for the expert analysis, and that they are in the hands of thoroughly responsible parties up to the date of analysis.

While *arsenic* may cause somewhat similar symptoms to those due to antimony, the stools are generally bloody from destruction of the gastro-intestinal mucous membrane by the drug. Rarely certain poisonous toadstools produce somewhat similar symptoms.

If an adult who has not eaten anything which could have pro-

duced a diarrhœa, as the result of irritation from bad food, is seized with profuse watery purging, with very little or no pain, and without nausea or vomiting, it is probable that he is suffering from the *acute nervous diarrhœa* which sometimes results from exposure to severe nervous strain. To illustrate the character of these cases the author may mention the fact that it is quite common for him to see medical students, exhausted by a long winter's work and anxious about their examinations, seized by an attack of profuse watery purging in the middle of the night preceding the examination of which they stand most in dread.

Care must be taken by the physician in all cases of sudden and profuse diarrhœa to which he is called to exclude the presence of renal disease, for purging may be an effort at elimination of effete materials, and its sudden arrest by drugs may induce uræmic convulsions or coma.

Sudden attacks of profuse watery diarrhœa in which the patient passes great quantities of liquid from the bowel, with or without pain in association therewith, may be due to *locomotor ataxia*, manifesting itself in an "intestinal crisis."

In cases of persistent or obstinate diarrhœa, serous or catarrhal, in which there is an excessive peristalsis which hurries the intestinal contents along so fast that the food cannot be properly digested, the physician should remember that *fissure of the anus* or some other source of irritation may be present in the lower bowel which produces reflex excitability of the nerves governing the bowel-movements. In other cases a stricture in a feeble, dilated rectum will cause retention of feces until irritation, tenesmus, and even loose mucous movements are produced.

If, instead of watery or serous movements, the patient is attacked by a more or less acute diarrhœa, accompanied by great pain and distention of the belly, and if there is marked tenderness on pressure over the transverse colon and mucus in the feces, which are not in very large quantities after the first few movements, there is probably present the condition known as *entero-colitis*, or inflammation of the ileum and colon. It is met with in both children and adults, and differs in its course from cholera morbus and cholera infantum very markedly. The pain is usually more constant, more aching, and less griping in character. Vomiting is not a constant feature, as it is in the watery choleraic diarrhœas, and the course is more subacute, the duration of the illness usually being from one to three weeks.

If food which is difficult of digestion has been eaten, it is passed, still undigested, from the bowel and is apt to be coated with mucus. Such a diarrhœa is called *lienteric diarrhœa.*

Not far removed from this type of cases are those of a more chronic character depending upon more grave and lasting alterations in the gastro-intestinal mucosa. As a rule, the greater part of the trouble exists in the colon, and more or less griping pain in the neighborhood, namely, in the upper umbilical area and left groin may be present before each movement. The abdomen is apt to be distended and quite tender on pressure, particularly in certain variable spots, and considerable loss of bodily weight is apt to ensue, chiefly from failure on the part of the digestive tube to absorb the food that is eaten. The movements are not markedly watery, but are usually unformed and about the consistency of oatmeal gruel or a little thicker. Flakes of mucus are often found in large amounts in the fecal matter, and the feces may be frothy or flaky as the result of fermentation. Blood and pus are very rarely seen in the movements of these cases, unless the blood escapes from an inflamed hemorrhoid. Sometimes, when these cases are very severe in character, the mucus takes the shape of long cord-like or worm-like strings, or even seems to be membranous in character. In other instances the feces, when formed, are passed in ribbon-shaped masses, either due to spasm of the muscular fibres of part of the lower bowel or to cicatricial contractions, due to the healing of old ulcerations. In very severe cases the condition of the intestine gradually advances from a *mild follicular entero-colitis* to one of actual deep ulceration, and under these circumstances blood and pus may be present in the movements. At such times the pain produced by the patient having a movement of the bowels, or by the passage of fecal matter over the ulcerated surface, may be intense, and the invalid will often state that the pain feels as if one spot in the gut were made more painful by the feces rubbing over it. Such cases often continue for years, and while some of them ultimately get well, others becomes chronic invalids from the slow changes in the intestinal walls. In this connection the diarrhœa of *tuberculosis* is not to be forgotten, depending, as it does, either upon the general infection or upon the development of ulcerations in the intestinal canal.

In some cases in which the patient after exposure to cold or wet is seized with violent pain in the epigastrium, and a feeling of weight in the rectum, a few loose movements, and then intense tenesmus

and bearing-down, with only a few drops of mucus in the way of a movement, the condition is one of acute rectal catarrh or *proctitis.*

The cases just named in the preceding paragraphs are to be separated from those in which there is *true dysentery.* Dysentery is a term very loosely applied, by the laity in particular, to any form of severe diarrhœa, particularly if there are blood and mucus in the movement. In reality the term dysentery should be limited to cases due to an infection and very apt to occur in epidemics. As Osler says, true dysentery is one of the four great epidemic diseases of the world.

Let us suppose that a patient is seized with diarrhœa and some pain in the belly, and with only a slight chill, or in other cases no chill may be present. The pain soon becomes more and more colicky and the stools are passed with ever-increasing bearing-down or tenesmus. The effort to empty the bowel, after it is in reality thoroughly emptied, results in agonizing bearing-down pains. Fever to the extent of from one to three degrees may be present. Thirst is excessive, the stomach is usually retentive, and the stools are first the ordinary bowel-contents, and then mucus, which may be blood-streaked. Soon the mucus becomes jelly-like in appearance and more thick and tenacious, and, finally, after several days it begins to look muco-purulent, and the stools are less frequent. Sometimes small bullet-like hard pieces of fecal matter are shot out of the rectum after severe straining. Recovery usually begins at from seven to ten days. The entire trouble seems to be in the large bowel, and particularly in the sigmoid flexure and rectum. Such are the symptoms of ordinary mild dysentery of hot climates or of summer weather in the temperate zone.

The severity of the disease is much greater in hot weather, and the prognosis is not good in severe cases coming on during an epidemic.

On the other hand, if the patient has an irregular diarrhœa, after, or during, a residence in tropical parts, which may or may not have a sudden onset, with moderate fever and considerable loss of flesh, and has moderate bellyache. which soon becomes much less, and if the stools as just described above become more and more fluid and the diarrhœa intermits, the physician should think of the case being probably one of so-called tropical dysentery, or *amœbic dysentery,* a condition of infection by the so-called amœbæ coli. The course of the disease is slow, lasting from six to twelve weeks,

and the death-rate is high. Convalescence is always very slow, and liver-abscess due to an hepatic infection by the amœbæ coli is very frequent. Sometimes abscess of the lung develops.

A positive diagnosis of this variety of dysentery is made by the discovery of the amœbæ in the stools. These micro-organisms possess active amœboid movements and are found in greater number when the diarrhœa is severe. They are to be sought for in the small gelatinous masses which are found in the feces. Sometimes the entire stool seems loaded with amœbæ, at other times only a most careful search will discover them. They are more refractive than the cells found in the feces, and contain numerous vacuoles, so numerous in some cases that the cells look very granular. These must not be mistaken for the compound granular bodies found in the feces. When they are active a division into an endosarc and an ectosarc can be discovered. Often red blood-cells will be found in the amœbæ.

Sometimes a diphtheritic or false membranous dysentery is developed in persons having chronic heart disease, and it has been seen as a result of acute croupous pneumonia. This is called *secondary diphtheritic dysentery*, and death generally results from exhaustion, only a suspicion of the intestinal condition having existed during life. Such a state is sometimes a complication of Bright's disease, probably owing to the irritation of the intestinal mucous membrane produced by the urea decomposing into carbonate of ammonium. In *acute primary dysentery of a diphtheritic character* the patient may rapidly pass into a typhoid state, and the case be diagnosed as one of typhoid fever with profuse diarrhœa. The discharges are the only means of separating the two conditions (enteric fever and diphtheritic dysentery), as they often are filled with blood and mucus in dysentery, a condition rarely seen in typhoid fever.

Dysentery may be confused with the diarrhœa sometimes produced by a malignant and ulcerating growth in the sigmoid flexure or rectum, but a physical examination will usually reveal the tumor, and the cachexia will aid in pointing to it as the cause.

Syphilitic ulceration of these parts may cause a somewhat similar train of symptoms. Again, it is by no means rare to meet with the passage of several muco-purulent movements each day in persons who have pulmonary gangrene or pulmonary tuberculosis, partly due to the swallowing of fetid sputum or tubercular ulceration of the bowels. Diarrhœa is also a symptom of septicæmia. Distantly

allied to this form of diarrhœa is that seen in persons who have dissected a putrid body (dissecting-room diarrhœa so-called).

While it does not fall to the lot of this book to discuss the symptoms of the affections of the rectum which are to be relieved by surgery, it is proper to speak briefly of the causes of *blood in the stools* in other states than dysentery. In inquiring as to the blood in the stools, we should ask whether it is mixed with the feces or is seen in streaks, and whether it passes in jets or not. If in jets, it will be found in the pan away from the fecal matter. We should ask as to the amount of blood and its color. If mixed with the feces, it probably results from a slow oozing from a hemorrhoid, or, if the feces are formed, from some leaking vessel in an ulcer high up in the sigmoid flexure. If in streaks, it probably comes from the wall of an ulcer which has been scraped by the fecal mass. If it is passed in jets, it probably comes from some vascular but small arterial tuft of vessels low down near the anus ; and, finally, if it is dark and tarry-looking, it is probably due to a leaking in the upper colon or ileum, whereas if light red in color it is from vessels in the rectum. Most commonly it is from hemorrhoids, or from an ulcerated mucous membrane covering a syphilitic deposit, or else a malignant growth. (See further on in this chapter.)

Finally, it is interesting to note that paroxysmal attacks of seromucous or bloody diarrhœa sometimes come on in case of exophthalmic goitre. Diarrhœa of a more or less severe type may come on in cases of hysteria, often associated with tremendous eructations of gas and rumbling in the stomach and bowels.

Fatty diarrhœa may ensue if feeble persons already suffering from irritable bowels take an excess of cod-liver oil, but in other cases it possesses great diagnostic importance. If associated with diabetes, it gives us reason to believe that there is some disease of the pancreas producing both the glycosuria and the lack of digestion of the fats. Sometimes in jaundice, however, fat is found in the stools owing to the lack of bile to emulsify it in the intestine.

The Feces. In this connection we naturally pass on to a discussion of the diagnostic indications of the feces. In the first place, it must be remembered that the quantity of the feces depends upon the quantity of the food, and again that the quantity varies with the character of the food, for if the food be such as to be bulky, yet contains little nutritive material, there will be a large residue to be passed out in the feces; whereas if the food be almost entirely com-

posed of materials which can be assimilated very little residue is left, and the feces are consequently smaller in bulk. Thus the cow eats a large bulk of food and passes large amounts of fecal matter, whereas the dog eats meat and passes very small amounts of fecal matter.

Again, it is not to be forgotten that many foods actually increase intestinal peristalsis, and so produce large and loose movements, as oatmeal and wheaten-grits or apples, while other foods, such as cheese, do the opposite. If the stools are large and copious and the food which the patient has taken is in reality not of a kind leaving a large residue in the bowel, the indication is that there is non-absorption of nutritive materials, with consequent wasting of the patient.

The consistency of the feces in health varies from a formed "stool" to a mushy condition; but in disease we have a liquid watery stool if the trouble be serous diarrhœa, and a pasty and slimy stool if it be due to a catarrhal state of the bowels. The passage of hard scybalous masses mixed with liquid indicates that the feces have become dried and hard in the sacculations of the colon, and are only passed out when they cause so much irritation as to produce diarrhœa. If the feces are in narrow bands or flattened ribbon-shapes, there is probably a stricture of the rectum, offering an obstruction to their passage. A mushy or semi-watery stool is often seen in typhoid fever. The odor of the stools depends very largely upon the food which is taken and upon the degree of fermentation present in the bowels. In nursing-children the stools often have a faintly sour odor, and in the diarrhœa of nurslings with acid fermentation there is an odor of the fatty acids. If the process is marked, this odor becomes actually foul, and in cholera infantum the stools have a musty, mousy odor. If malignant growth of the bowel is present, the odor is fetid, as it is also in gangrene of the intestine. Sulphur when taken internally causes a very offensive stool, owing to the sulphuretted hydrogen gas which is developed in the bowel.

The color of the stools is of great diagnostic importance in several conditions. In health the feces should be brown or brownish-black, the color being partly due to the food, but chiefly to the bile (hydrobilirubin). Certain fruits render the stools dark in color, and drugs, such as iron and bismuth, do likewise, and hæmatoxylon often makes them look red.

In the stools of persons living on a pure milk-diet we usually find

little color comparatively. Again, in cases of jaundice, phosphorus-poisoning, and acute yellow atrophy of the liver, the stools are very light in color, owing to their lack of biliary coloring. They are also apt to be very light in chronic lead-poisoning.

Bilious stools are either golden yellow, greenish, or reddish in hue, and if the flow of bile is profuse, they are apt to be watery. Greenish stools looking as if they contained chopped spinach are, however, a peculiarity of the diarrhœa of fermentation, particularly in infants, the color being due to a color-forming micro-organism.

If the stools are well mixed with mucus, the catarrhal process probably exists in the ileum; but if they consist of hard masses of feces coated with mucus, the disease is probably a colitis.

Bloody stools are most commonly due to hemorrhoids which are eroded. The blood may be bright if the hemorrhoid be a small arterial bunch, or more dark and grumous if slow oozing has gone on for some time prior to the movement. As a rule, the brighter the blood in the stool the nearer its source is to the anus, and the darker the blood the higher its source in the bowel. Thus, if the stools are tarry-looking, the blood is almost certainly from the small intestine, and probably arises from a duodenal or other ulcer or from carcinoma of the stomach or bowel; while if it is only somewhat changed in appearance, it may be due to an ulcer or ulcerated morbid growth in the colon. Sometimes, however, where the hemorrhage from the ileum is very profuse, as in typhoid fever, the blood comes from the anus only slightly changed in appearance. If blood is suspected to be present, we can confirm its presence by finding the corpuscles by the microscope or by testing the feces for hæmin. (See chapter on Vomiting.)

Stools containing pus may receive this material from the surfaces of ulcers, but usually the source of the purulent matter, if it is present in large amount, is an abscess which has ruptured into the bowel, as, for example, in peri-rectal abscess.

Finally, we may find gallstones in the stools, which, if they are passed soon after escape into the bowel, are found to be faceted. Stools which are being searched for gallstones should be washed through a sieve in such a way as to catch the stone and let the fecal matter through. The intrahepatic gallstone is not faceted and crumbles easily.

Very rarely a portion of the bowel sloughs away, and yet recovery takes place. This is seen sometimes in intussusception.

Aside from the character of the stools themselves, we often search

for the cause of an ailment in the passages, either for foreign bodies, such as pebbles or pins, or for *intestinal parasites* (worms). Sometimes worms may exist for long periods of time in the bowel without causing any symptoms, and, again, in children in particular, they cause great systemic disturbance by producing disorder of the digestion or reflex irritation.

Under the name of tapeworm or cestodes, we find in the intestine, and often in the stools, a parasite occurring in segments which are flat and ribbon-like, and usually from a quarter to one-half inch in length. The worm itself may be several yards long. Its head is small, and it maintains its hold on the bowel by its head. The segments are usually broken off one by one and escape in the stools, and the stools also contain the ova or eggs of the parasite, which are developed in each segment, which also possesses male and female organs.

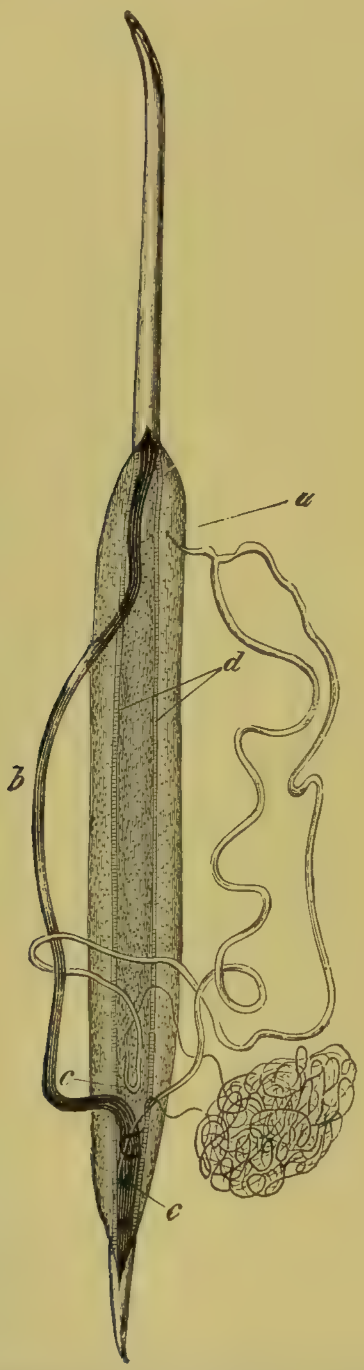
According to the shape of the head and the size of the worm and the source of infection, we divide tapeworms into three classes : the tænia solium, the tænia mediocanellata, and the bothriocephalus latus.

If the patient passes a worm of from one to three yards in length, the head of which is about the size of a pin-head and glistening gray in appearance, the rest of the worm being yellowish-white, and if upon the head can be seen four pigmented suckers surrounded by a crown of hooks, that worm is a tænia solium, and is probably derived by the patient from raw or uncooked pork. The eggs of the tænia solium must be sought for by a microscope. They are round and covered by a hard shell, which breaks upon pressure into small fragments. In the shells may be found a few hooklets. These eggs are passed out in the feces by the host, and are then swallowed by the pig, in whose muscles the hooklets migrate and form cysts. In these cysts the hooklets develop, and when a man eats the meat raw they enter his intestine, attach themselves, and from them a tapeworm is developed.

If the worm is from four to five yards long and the segments after leaving the anus have motile powers, and if the head is larger than that of tænia solium and devoid of hooklets about the suckers on its head, it is probably the tænia mediocanellata or saginata. The egg is slightly larger than that of the solium. This worm usually comes from eating raw beef. The bothriocephalus latus is the largest of all tapeworms, often reaching seven to eight yards in length. It

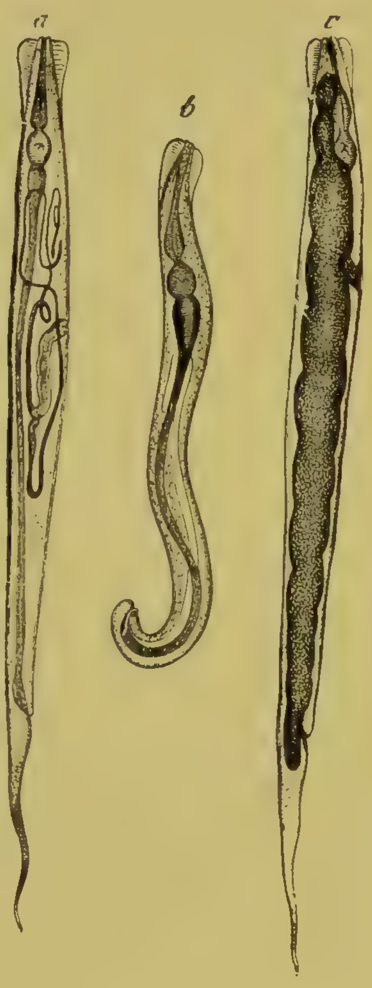
has a long head with two long, narrow suckers. The eggs are oval, very large, and the shell is light-brown in color and very easily broken. This parasite is not common in America, but is a very

FIG. 171.



Ascaris lumbricoides, dissected and walls thrown back. (HELLER.) *a.* Genital orifice. *b.* Intestine. *c.* Oviducts. *d.* Longitudinal band. *e.* Ovaries.

FIG. 172.



Oxyuris vermicularis, magnified. (PAYNE.) *a.* Young female. *b.* Male. *c.* Mature female, full of eggs.

frequent cause of profound anæmia in the persons whom it infects. Its joints are only rarely thrown off, so its presence is often overlooked, and this renders the search for the eggs very important in severe anæmia with no ascribable cause. This worm is usually derived from fish. A worm which is comparatively rare is the *tænia*

FIG. 173.

Trichocephalus dispar, natural size. (PAYNE.)
a. Female. *b.* Male.

FIG. 174.

Ankylostomum duodenale, magnified. (BRISTOWE.)
a. Female.
b. Male.

cucumerina, which has a head with sixty hooks. It infects dogs, cats, and sometimes children.

A round worm, looking like an ordinary earthworm, appears sometimes in the stools, and is called ascaris lumbricoides. It is sometimes vomited, and, rarely, causes trouble by crawling into and blocking the common biliary duct.

Fine thread-like worms inhabiting the rectum are the oxyuris vermicularis.

A very important diagnostic find in the feces is a worm, looking very much like the thread-worm, but somewhat larger, which inhabits the duodenum. It is called the ankylostomum duodenale. The importance of finding it lies in the fact that it produces the most profound and acute anæmia by sucking blood from the intestinal wall. The worms are usually only found after a vermifuge is taken, but the eggs are always present as unsymmetrical, thickly covered segmented globules. If the stools containing the eggs be set aside in a warm place, the embryos can be seen to develop in the eggs. Bloody stools may be due to the presence of this parasite.

The so-called whip-worm, or trichocephalus dispar, is a fine thread-worm without any medical interest.

PART II.

THE MANIFESTATION OF DISEASE BY SYMPTOMS.

CHAPTER I.

FEVER AND SUBNORMAL TEMPERATURES.

The methods of taking the temperature—The significance of fever—The febrile movements of various diseases.

FEVER is that state of the human body in which its temperature is raised above the normal limit, or 98.8° F. Hyperpyrexia is a term applied to a febrile movement in which the temperature rises as high as 106° F., and cases are on record of a temperature of 115° or even more.

The method of taking the temperature consists in placing a self-registering clinical thermometer in the mouth under the side of the tongue, the lips being then closed tightly about its stem; or of inserting it in the axilla, the hand and arm being then placed across the patient's chest, or epigastrium, so as to cause the axillary tissues to be in close contact with the bulb of the thermometer. Before the thermometer is placed in the axilla this space should be carefully wiped dry, since if perspiration is present its evaporation will so chill the thermometer that a false record will be made by the index. Sometimes the temperature of the patient is taken by inserting the thermometer into the rectum, and, if this is done, the bulb should be passed well inside the external sphincter. Rarely the temperature is taken in the vagina.

The precautions to be taken in all cases in which a thermometer is used, in addition to those named, is to have a thermometer which is accurate, and to be sure that there is no acute or chronic inflammatory process present which will produce local heat, and so give an erroneous impression as to the actual temperature of the entire body. This is particularly apt to be the case in diseases of the

mouth in children, thus stomatitis may raise the local temperature from one to two degrees. Hot liquids, if taken into the mouth just previous to or during the time at which the thermometer is inserted, will so raise the temperature of the local tissues as to make the thermometer register several degrees above normal, and a similar effect may be produced by cold liquids or ice held in the mouth. This subject has recently been studied by Lazarus-Barlow, who asserts that the effects of hot objects taken into the mouth last much longer than do those produced by cold, and that a mouth-temperature should never be taken within one hour of the time that any hot food is ingested. He even shows that holding the mouth open for some time renders a true estimate of the body-heat impossible, and advises that the temperature shall never be taken in the mouth if it is possible to take it elsewhere.

The significance of fever is great. It always shows the presence of an ailment sufficiently severe to make it wise for the physician to order the patient to bed till the fever abates or until he can surely determine its cause. The significance of a raised bodily temperature from a physiological point of view is that the nervous centres governing heat-production and heat-dissipation are disturbed by some substance circulating in the blood or by reflex irritation, or perhaps by both. The danger of high fever is that it may cause coagulation of the protoplasm of the heart or vital centres in the base of the brain, but the danger of ordinary febrile temperatures has been greatly exaggerated. Indeed, in some cases moderate fever probably aids the body in throwing off or, rather, conquering the disease which has attacked it, in three ways, namely, by producing a temperature less favorable to the growth of certain disease-germs than is the bodily temperature in health; by increasing cellular activity it may increase phagocytosis and the development of antitoxic materials; and, finally, by virtue of the increased temperature the effects of poisons may be rendered *nil*. This is the case, for example, in regard to digitalis, which will rarely produce its ordinary effects on the heart when well-marked fever is present. Another point of importance in this connection is this, namely, that the duration of fever has more to do with its importance as a symptom than has its degree, for a temperature of 105° for a few hours may be borne with immunity, whereas one of 103° for many days cannot fail to produce evil effects.

Febrile movements are generally associated with a dry, hot skin,

but sometimes with a cold, wet skin. The latter condition is of evil significance as a rule, and must be overcome if possible.

Fever in children does not possess nearly as much significance as it does in adults, for children often develop high temperatures from slight causes and have speedy recoveries. The balance of their heat-mechanism is easily upset. The older the patient the greater the significance of fever, and a rise of two or three degrees in a man of sixty years is more alarming than one of four or five degrees in a child of five or six years.

When fever is not due to some distinct pathological change in some part of the body, generally of an inflammatory kind, it may arise from mild irritation of a mucous membrane so that a catarrhal condition is set up. Such fevers are seen in cases of mild gastro-intestinal catarrh in children after the ingestion of bad food or exposure to cold, and apparently arise as the result of the reflex irritation produced by difficult teething. (See chapter on the Tongue.) In many instances, however, the fever of dentition depends upon a more or less closely related, but overlooked, gastric catarrh. Sometimes after a urethral sound or catheter has been passed in a man, in the course of a few minutes or hours he develops a severe chill, followed by a fever which may be quite high, but does not last long.

Fever in Infectious Diseases. Nearly all infectious diseases are ushered in by the development of fever of greater or less degree, and this is particularly true of the exanthemata. In *typhoid fever* the febrile movement is very characteristic in some cases, although in many instances it does not follow the description laid down in text-books. After several days of general wretchedness the patient develops a slight fever of from 100° in the morning to 101° at night, and this temperature progressively rises so that the next morning it may be 101° and that night 102°, the next morning 102°, that night 103°, and so on until the morning temperature may be 103° and the evening temperature 104° or rarely 105°. The fever usually reaches its acme by the end of the first week or ten days, and then for another week remains almost unchanged, there being a morning fall and evening rise of an almost equal extent. Toward the end of the third week, or sometimes earlier or later, according to the severity of the attack, the morning remissions become more marked and then the evening rises fail to reach their former height. Often these marked morning remissions

are the first indication of the tendency to recovery. Very high evening temperatures are indicative of a severe attack, but are not so indicative of serious illness as are high temperatures in the morning. After the third week, in a moderately severe case, the temperature falls gradually till by the twenty-eighth day it usually reaches the normal. In very rare cases the temperature speedily reaches its acme at the very beginning of the disease and then passes through the course already described. Such cases are generally prolonged, but may in some instances end by the fourteenth day.

Sudden falls of temperature during the course of typhoid fever are nearly always of grave import. The most common cause of such a sudden fall is an intestinal hemorrhage, and the fall may occur sometimes before the blood appears in the stools. In other cases it is an evidence of intestinal perforation. The other causes of a sudden fall are severe nose-bleed, or hemorrhage of any form; as, for example, that occurring in connection with abortion in a female patient. Sometimes, too, without any of these causes, the temperature falls very rapidly, and the patient goes into collapse. Such cases are very grave and the prognosis unfavorable.

FIG. 175.

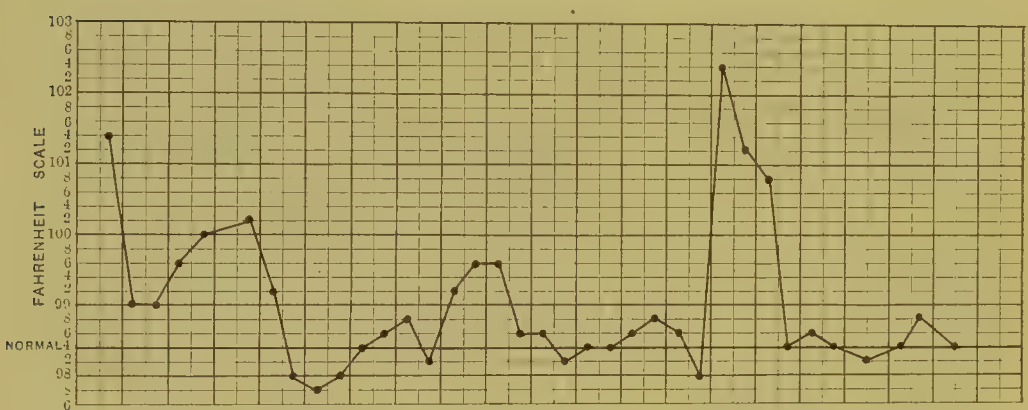


Chart showing recrudescence of fever in a case of typhoid fever.

A recrudescence or return of the fever, in which it rises quite rapidly to a point as high or higher than at any time during the attack, occurs in some persons who, during the stage of convalescence from typhoid fever, take solid food too soon, or are excited by the visit of a friend. Such rises are but temporary. (Fig. 175.) More rarely, as a result of getting out of bed, or bad feeding, or other causes, a true relapse takes place, and the disease runs a second course, which is

ward course. The febrile movement is repeated at intervals, ranging from one to seven days or even at longer intervals than this. If the attacks occur daily, they are called quotidian, and this is due to infection by two sets of tertian parasites which segment on alternate

FIG. 179.

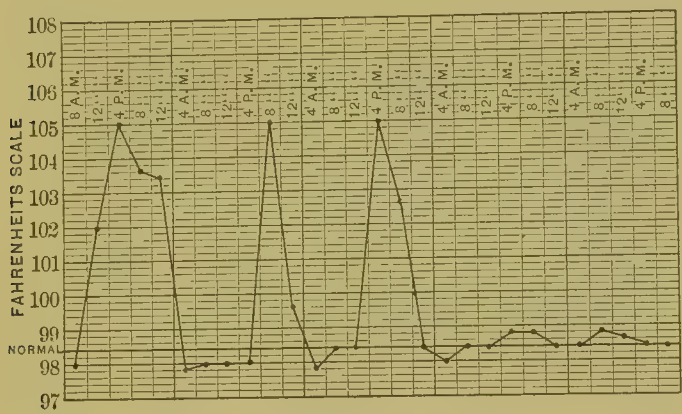


Chart showing daily paroxysm due to double tertian infection. One set of parasites segmented at 4 P.M. and the second set at 8 P.M. Paroxysm stopped by quinine on fourth day.

days, or it may be due to infection with three sets of quartan parasites. If the attacks occur every other day, they are called tertian (Fig. 180); if on the third day, quartan; if on the fourth, quintan. If two attacks come on the same day, it is called double quotidian.

FIG. 180.

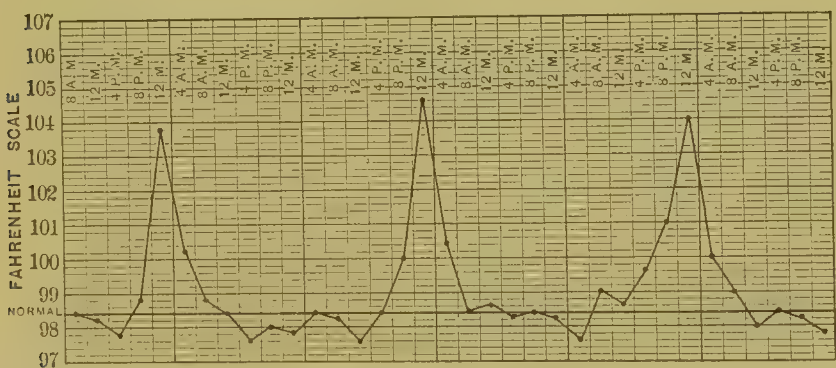


Chart showing paroxysms of tertian fever, the segmentation of the organism occurring at about twelve o'clock each day.

Another point of importance in connection with malarial attacks is that they of ten occur earlier each day by anhour or more. Rarely, they are delayed.

Intermittent malarial fever is to be separated from other intermitting fevers by a number of facts. First, the presence of the mala-

rial organism in the blood at the time of the attack, or evidences of its presence at other times. (See chapter on Blood.) Second, by the history of exposure to malarial influences. Third, by the marked effect for good on malarial fever produced by the administration of quinine. Care must always be taken that the intermitting fever of the various forms of sepsis are not diagnosed as malarial intermittent fever. The most common error of this character is the making of a diagnosis of irregular malarial intermittent, because chills, fever, and sweat appear every evening, when, in reality, the real cause is an undiscovered pulmonary or abdominal tuberculosis. Again, acute ulcerative endocarditis and purulent phlebitis may cause similar symptoms, as may also hepatic abscess, inpaction of gallstones, with suppurative cholangitis, causing the so-called Charcot's fever (see below). The absence of a history of malarial exposure, the possible presence of a cough, and the discovery of a tubercular lesion in the chest or abdomen by careful physical examination will aid in deciding that the fever is tubercular and not malarial in origin. (See chapter on Chest and on Abdomen.)

In *ulcerative endocarditis* the temperature-curve may exactly resemble intermittent malarial fever, but in many instances the presence of an external wound, acute sepsis in some part of the body, or the presence of the puerperium will reveal the source of an infection. (Fig. 181.) In the typhoid type of ulcerative endocarditis the profound asthenia and general prostration will separate the diseases even if the temperature-chart be unequal. In this form the febrile movement is rarely typically intermittent. The crucial test of the differential diagnosis lies in an examination of the heart, in which a murmur may be heard in some but not in all cases, unless there has already been some grave valvular mischief. The cardiac feebleness and asthenia, on the one hand, and the result of the blood-examination, on the other, aid the diagnosis. The duration of the case is not of much value in making a diagnosis, for cases of ulcerative endocarditis have lasted from two days to more than a year. Rarely it lasts more than six weeks. Death usually occurs in ulcerative endocarditis, unless there has been previously present chronic endocarditis, in which case recovery may rarely occur.

The discovery of some spot showing a *phlebitis* may point to this cause for intermittent fever.

The fever of *catarrhal* or *suppurative cholangitis* often closely resembles intermittent fever, but the presence of hepatic symptoms,

is *Weil's disease.* In this condition the fever runs a remitting course, is associated with jaundice and swelling of the liver and spleen, and the stools may be clay-colored. There is one important point of difference between malarial remittent fever and Weil's disease, namely, that in the latter gastro-intestinal symptoms are nearly always wanting or are mild, whereas in the former they are apt to be very severe. Usually the fever of Weil's disease ceases by the end of two weeks or earlier. It is probably an infectious jaundice.

In *dengue,* a disease seen most commonly in epidemics in certain parts of the Southern United States, the patient, after suffering from violent aching pains in the body and limbs, swelling of the joints, and the development of a variable rash on the chest, develops an active fever, which lasts with the pain till the fifth day, when both the pain and fever decrease or cease, and then often return with equal force. These facts, combined with the fact that it is an epidemic disease, separate it from malarial fever.

The fever of *yellow fever* is rarely over 103° or 104°, and is one of the milder symptoms of the disease, but it possesses this peculiarity, namely, that after the lapse of from twelve hours to several days there is a marked remission of the fever and all the other symptoms, and from this time on the patient may get well, or after a few hours this calm stage is followed by the true violent symptoms of the disease, such as black vomit, tarry stools, jaundice, and hemorrhages from the mucous membranes. Generally the full course of disease to convalescence or death is run in about one week.

There is only one other disease which can be readily confused with yellow fever, namely, bilious remittent fever, and a case of the latter disease occurring during an epidemic of yellow fever can hardly fail to be incorrectly diagnosed. In the absence of an epidemic, however, the probabilities of the case being bilious remittent fever are very great, and the presence of bilious vomiting rather than that of blood, the characteristic temperature-chart, and, above all, the presence of a history of malarial exposure and of the signs of malarial infection in the blood, with the partial control of the symptoms by quinine in certain stages of remittent fever, point to the malarial disease rather than to yellow fever.

Just as in yellow fever, so in spotted fever or *cerebro-spinal meningitis* of an epidemic form, the fever itself is one of the least important symptoms, for, aside from the fact that it is apt to be irregular and intermitting, it is rarely very high, as compared with

the violent cerebro-spinal symptoms, the rigidity of the back of the neck, the headache, convulsions, and vomiting. The presence of these symptoms in an epidemic does more to confirm a diagnosis than the febrile movement. In some cases of spotted fever, however, of a very grave type, the fever becomes a hyperpyrexia, but in cases tending toward recovery the temperature usually begins to fall by lysis before any moderation in the other symptoms is manifested.

Even in the presence of an epidemic it should never be forgotten that middle-ear disease often causes marked meningeal symptoms, and that croupous pneumonia often produces a similar train of manifestations, probably by infection with its particular micro-organism. The possibility of tubercular infection should cause the physician to examine the patient carefully for signs of tubercular disease in other parts of the body from which infection might arise, as, for example, the lungs.

Fever due to *septicæmia* may produce a temperature-chart which closely resembles that of enteric fever, but it generally possesses one characteristic which, in the face of other symptoms suggesting sepsis, is of great importance, namely, the extraordinary rises and falls from normal to 105° or 106°, and from that point even to a subnormal degree within a very few hours, so that the lines on the chart pass up and down in long sweeps. These sweeps are even more sharp and sudden than in an intermittent malarial fever, and their cause is determined by the discovery of some septic process in some part of the body. The presence of such a chart, in association with dull or violent headache, delirium, vertigo, and vomiting independent of taking food, would point to cerebral abscess, particularly if a history of injury could be obtained.

A somewhat similar chart to this may occur in connection with cases of *active pulmonary tuberculosis*, when the lesions are well developed and septic absorption is active; but usually in the hectic fever of phthisis we have a normal morning temperature, with a rise from two to three degrees, or even more, toward night. (Fig. 182.) This symptom of fever in any form occurring in a person with suspiciously "weak lungs" should cause the physician to be confident that he has overlooked some focus which another careful examination may discover, and it possesses another important diagnostic significance, namely, that the more active the febrile movement in phthisis pulmonalis the more active the disease process, and less active the fever the less active the process. Fever may, however,

be almost entirely absent in some tubercular cases with diseased lungs.

The febrile movement of *acute miliary tuberculosis* has nothing characteristic about it, except that in some cases it may closely resemble that of typhoid fever, and if the physician does not carefully examine the case an erroneous diagnosis may be reached. If, however, the disease involve the meninges of the brain, a hyperpyrexia may be developed, and death speedily occurs in such cases, either in the fever or in a sudden collapse. The peculiar dyspnœa, the cyanosis, the profuse sweats, and the diffuse pulmonary signs render a diagnosis of acute miliary tuberculosis possible in some cases.

FIG. 182.

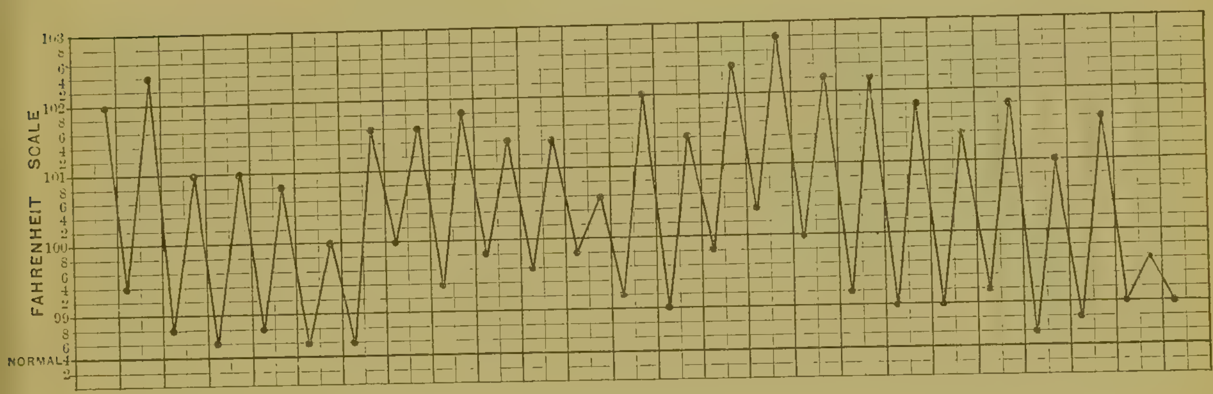


Chart of a case of pulmonary tuberculosis, showing rising and falling of the temperature morning and night.

When fever is associated with marked catarrhal symptoms, chiefly of the bronchial tubes and upper respiratory tract, with sneezing, lassitude, pains in the back and limbs, and excessive cough, the fever rising as high as 104° or 105° in severe cases and then falling almost to normal, we may have before us *influenza* or catarrhal fever, either of the sporadic or epidemic form. In this condition there may be in severe cases great prostration and cardiac failure or vomiting and diarrhœa. The febrile movement is of the most irregular type, even when some grave complication, such as severe bronchitis or pneumonia, comes on, although croupous pneumonia rarely occurs as a complication of "la grippe."

The respiratory symptoms just described are also seen frequently, in association with moderate fever, in "*Hay Fever*," that condition seen in susceptible persons during haying-season or late in the summer.

The fever of *pneumonia* of the croupous type runs a very typical course in uncomplicated cases. Following a more or less severe chill, the fever quickly mounts to the high point of 103° or 104°, or even more than this. (Fig. 183.) For the next few days, if not modified by antipyretics and the use of cold, the fever remains high; but there may be temporary remissions which look as if crisis was about to be established, when in reality they are followed at once by a return of the fever (pseudo-crises). Finally, in the majority of cases of croupous pneumonia the temperature suddenly falls by crisis on the seventh to ninth day (Fig. 184) and convalescence is established, although the sudden fall of fever may throw the patient into dangerous collapse. Sometimes this convalescence is broken by brief and slight febrile movements. If the case has been prolonged, or of the typhoid type, the fever may end by lysis.

FIG. 183.

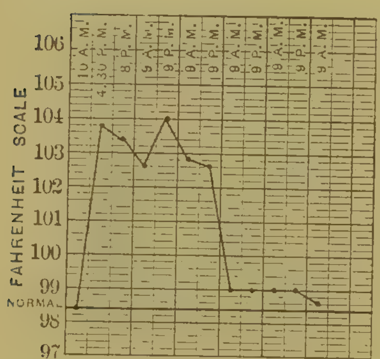


Chart of a case of croupous pneumonia, showing primary rise of temperature to 103.4° and crisis occurring as early as the third day.

FIG. 184.

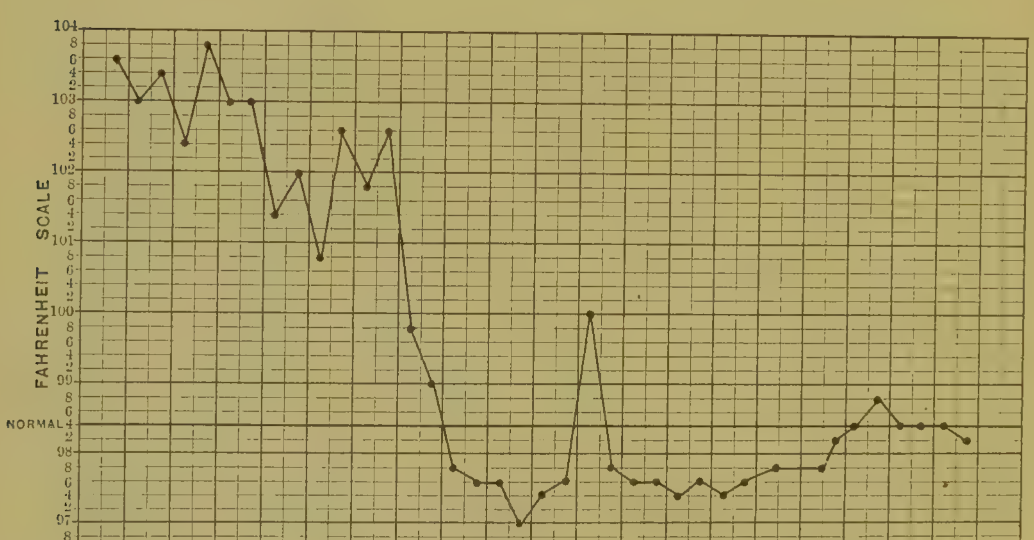


Chart of a case of croupous pneumonia, with crisis on the seventh day; admitted to author's wards on second day of illness.

It is to be remembered that the fever of *catarrhal pneumonia* is rarely as high as in the croupous form, 101°–103°, and ends by lysis, not by crisis. (See chapter on the Chest.)

The fever of *acute bronchitis* possesses no peculiarities over that of other acute inflammations.

It is not proper to leave the subject of fever due to the various infectious diseases without calling attention to that due to *syphilis* in the secondary period of its course. With the onset of the roseola or other skin-lesion a fever, more or less marked, is nearly always present and is often preceded by chilly sensations and general malaise. This febrile movement may then follow one of three courses: it may never rise above 101°, and proceed as does a simple continued fever, with slight morning remissions and evening exacerbations; or it may be as remittent as is a malarial remittent fever; or, again, it may resemble a malarial intermittent, rising to a high point and then falling almost to the normal. Phillips, of London, has reported a case of syphilitic fever in which this febrile movement lasted for weeks, and, after being treated by quinine as a supposed tertian ague, ended at once under antisyphilitic medication. (See chapter on Skin Eruptions.)

In *anthrax* (splenic fever) the temperature rises rapidly and becomes very high, and in the course of from three to five days becomes subnormal, when death occurs. The history of exposure to possibly infected hides or hair, the early development of a papule, vesicle, and pustule, surrounded by brawny swelling and enlargement of the neighboring lymph-glands, renders the diagnosis easy; but if any doubt exists, it can be promptly dispelled by a microscopical examination of the fluid from the pustule, when, if the disease be anthrax, the anthrax bacilli will be found. (See chapter on Skin.)

Fever with a vesicular eruption about the lips and on the mucous membrane of the mouth accompanied by disorder of the stomach and bowels may be due, particularly in children, to infection by the milk of cows suffering from *foot-and-mouth disease* or epidemic stomatitis. The prognosis is generally exceedingly unfavorable.

In *cholera Asiatica* during the stage of collapse the surface is very cold, but the rectal temperature may be found as high as 103° or 104°.

In *cholera infantum*, which is a form of gastro-intestinal irritation often produced by infected milk, there may be fever amounting to 102° or 103°, and not uncommonly much higher, even to 107° or 108° in fatal cases. The diarrhœa and obstinate vomiting, the age of the patient, the season of the year (usually hot weather), and the profound wasting, all complete the array of facts necessary for diag-

nosis. It is important to remember in these cases that the skin may feel cold and clammy even when the rectal temperature is very high.

The febrile movement associated with the progress of *acute parenchymatous nephritis* may or may not be preceded by a chill. The temperature may rise to from 100° to 104°, but the course of the fever itself is of no diagnostic import. The pulse, pain in the back, headache, perhaps drowsiness and coma, and the diminished urinary secretion, bloody urine, and albuminuria, render the diagnosis easy and the cause of the fever evident. Very marked fever up to 104° or 105° may develop in the early course of acute infectious tonsillitis or in suppurative tonsillitis.

The fever seen in most cases of *tetanus* is very moderate, but it is subject to excessive fluctuations, and in cases approaching a fatal ending may reach 110°.

The fever occurring in *acute appendicitis* is a very unreliable symptom, notwithstanding assertions to the contrary. It rarely rises above 101° or 103° and sometimes not above 100°. Even in those cases in which the peritoneum has become involved by the inflammation the fever may not be marked, particularly if the peritonitis is septic. In other words, the presence of fever in association with pain in the right iliac region is a positive sign of some irritative or inflammatory process; but if the physician excludes appendicitis on the ground that fever is not present, he may make a serious mistake.

The fever of ordinary cases of *acute articular rheumatism* is usually moderate, rarely exceeds 103°, and possesses no typical characteristics; but in very severe forms of the disease with cerebral manifestations, a rheumatic hyperpyrexia may be developed, when, with delirium, convulsions, and cyanosis, the fever rises to 106° and even to 108°, after which death often ensues. The history of previous attacks of articular rheumatism, the hot, swollen joint or joints (usually the large ones), and the successive invasion of other joints as the ones first affected get well, point to the correct diagnosis. It must not be forgotten, however, that gonorrhœal and other forms of septic arthritis occur with febrile movement. Pyæmia, osteomyelitis, and purpura also may produce a fever with swelling of the joints. (See chapter on Legs and Feet.)

When a person, previously afebrile, during hot weather or when exposed to artificial heat in excess, is attacked by unconsciousness, convulsions, and very high fever, he is probably suffering from

thermic fever or heat-stroke. Theoretically similar symptoms might be caused by a lesion due to embolism or hemorrhage in the neighborhood of the pons Varolii, but this is very rare. (See chapter on Monoplegia and Face and Head.) The fever in sunstroke may rise as high as 110° to 112° or even more; the skin is hot and dry, or more rarely cold and moist with sweat, but, even if this is the case, the rectal temperature will be found hyperpyretic.

A great rise of temperature (110° to 112°) often occurs after injuries to the cervical region of the spinal cord.

Fever of considerable degree may be met with in cases suffering from hysteria, acute febrile neuritis, infantile spinal paralysis, apoplexy, and acute myelitis.

Temperatures as high as 106° have often been reported as occurring in *hysteria*, but in a certain number of cases these records are really fictitious and produced by some trick with the thermometer. Only the rectal temperature, taken while the physician is present, should be relied upon in such cases.

The rapid development of fever, pain in the back and limbs, and particularly in the nerve-trunks, the temperature soon reaching 103° or 104°, may be due to an attack of *acute multiple neuritis*, and the history that the illness has followed exposure to cold and wet may, on the one hand, make the physician believe that his case is suffering from rheumatism or influenza, or on the other, in the absence of such a history, from the early stages of one of the infectious diseases. The early appearance of tingling, numbness, loss of power, and wasting of the muscles soon decides the diagnosis in favor of neuritis. The nervous disease which most closely resembles acute febrile neuritis is Landry's paralysis, and a differential diagnosis may be difficult; but in neuritis there are loss of sensation, muscular wasting, signs of degeneration, and fever, whereas in Landry's paralysis all these are wanting, excepting the sensory symptoms, which in both diseases may be similar. The predominant symptoms of Landry's paralysis are paralysis and loss of reflexes. (See chapter on the Legs and Feet.)

The prognosis of the severe form of febrile neuritis is grave, as death may ensue from respiratory paralysis.

The fever of *infantile spinal paralysis* (anterior poliomyelitis) often is the chief symptom ushering in the disease, and rises to 104° or even 105° in some cases. There may be convulsions, headache, and twitching of the muscles, and, after the acute attack has passed off,

loss of power is speedily discovered in several muscles of one limb as a rule. (See chapter on Legs and Feet.)

Fever at first amounting to only one or two degrees, but afterward rising as high as 104°, associated with numbness and weakness of the legs and loss of reflexes, followed by paraplegia, may be present as a symptom of *acute myelitis*, traumatic or otherwise. (See chapter on Legs and Feet, part on Paraplegia.)

A rise of several degrees of fever may come on after an epileptic convulsion.

A febrile attack not rarely seen in children, yet not readily placed under the heading of any given disease, has been described by Donkin and Goodhart, and the writer has also met with it quite frequently. This condition has been called *gastro-pulmonary fever;* but as either pulmonary or gastric signs may be absent, this term does not apply to all cases. A previously healthy child is suddenly seized with marked fever, rapid respirations, and râles may be heard in its chest. There are often vomiting, headache, and drowsiness, with recovery taking place in several days. Often these attacks are associated with gastric catarrh, but sometimes this state is not present. Donkin states, and it is the writer's own experience, that they are apt to be produced by fright or excitement. In a case of the writer's the sight of an angry skunk attacking a pet dog produced a violent attack.

Subnormal temperature of the body is seen as the result of any profound nervous shock, as after an accident or surgical operation, or prolonged anæsthetization. It occurs, too, at the ending of the fever of croupous pneumonia and other febrile movements ending by crisis. It is also seen in severe cholera morbus and cholera Asiatica and sometimes in cholera infantum, and often is present either in the early part of the cold stage of intermittent malarial attacks or more commonly after the fever of the attack has fallen. Subnormal temperatures are also seen in some cases of confusional insanity, and of tubercular meningitis and hysteria.

An important variety of subnormal temperature is that seen in the form of heat-stroke called heat-exhaustion, when, in place of fever, a condition of collapse is induced. Severe injury to the dorsal region of the spinal cord often produces a great fall of temperature.

CHAPTER II.

HEADACHE AND VERTIGO.

The causes of headache—Digestive headache—Headaches due to the eyes—Headaches due to cerebral growths and abscess—Headaches due to syphilis—Headaches complicating acute diseases.

HEADACHE is, of course, always a symptom and never a disease, and it arises from such widely different causes that it is impossible in this book to discuss all of them. Only the more common conditions resulting in its development can be considered, more particularly in relation to its diagnostic significance in serious pathological states. The most common cause of headache is probably disorder in the function of the digestive apparatus, the next most common cause is eye-strain in its various forms, and the third is nervous exhaustion or neurasthenia with or without associated anæmia. These may be all considered as perversions of function causing headache—that is, the pain in the head may be termed a functional headache. Less frequently, but far more important from a diagnostic standpoint, is the headache seen in persons suffering from renal disease, brain-tumor, and meningitis in its various forms. The remaining causes of headache in both of these classes are numerous, and some of them will be considered later; but the most important of the first class are the headaches of the gouty or rheumatic, and of the second class those of cranial periostitis, middle-ear disease, and acute inflammations of the eye or in the jaw.

Headaches depending upon disturbance *of the digestive system* are nearly always accompanied by evidences of such disorder, consisting in gastric or intestinal distress, belching, hiccoughing or vomiting, or even by diarrhœa. Often there is a distinct history of the ingestion of indigestible food or digestion-disturbing drink, but in other cases exposure to cold so congests the abdominal viscera that catarrh of the stomach and bowels is induced, and with it congestion of the liver followed by jaundice. The headache of disturbed digestion is nearly always frontal, and in many cases congestive to such an extent that the face may be flushed, or at least the intracranial circu-

lation is so disturbed that the patient is unable to lower the head, because such a posture increases the pain. Such cases are relieved by hot foot-baths, which relieve the congestion of the head; nearly always by the act of vomiting, which should be induced if need be by an emetic or by putting the finger in the back of the throat. Vomiting makes such headaches very much worse for a time, owing to the congestion of the head in the efforts at vomiting, and this is an important point in diagnosis, for in renal disease or some other states the vomiting is sometimes so easily performed that no straining ensues.

That disturbances of the digestive tube are capable of altering the intracranial circulation is proved by numerous facts. Thus Brunton quotes the experiments of Ludwig and Dogiel, who showed that moving the intestines by the finger introduced through an abdominal incision caused a great increase in the flow of blood through the carotid arteries.

Headache due to disorder of the digestion rarely ensues immediately after food is taken, since some time must elapse before the ingested material becomes changed into an irritating or toxic mass by fermentation or putrefactive processes. As a consequence several hours or even a day may pass without any discomfort in the head, after which time the full force of the headache develops. The headaches of indigestion are, however, characterized by two important facts, viz., that they are not constant, and, second, that they are often relieved or prevented by the use of a purgative, even if constipation has not been present. Such headaches are very apt to be pulsating and accompanied by great nausea. Sometimes such a headache takes a form called migraine or hemicrania, a condition in which the pain is chiefly, if not entirely, unilateral, and there is associated with the pain early and more or less persistent hemianopsia. It is to be remembered, however, that in some cases of hemicrania of nervous origin the sickness at the stomach seems to be secondary to the severe pain in the head.

Headaches resulting from digestive disturbance do not always depend entirely upon irritation of the stomach and bowel with reflex disturbance of the circulation and sensory nerves of the head, but upon the absorption of poisonous substances formed in the digestive tube. These poisons are usually formed only to be destroyed by the liver, or are developed in too small quantities to have any effect; but no sooner do congestion of the liver and deficient biliary secretion

ensue than they are formed in large amounts, and enter the general blood-stream, owing to the absence of antiseptic bile and the coincident or consequent constipation. As a result, we see very violent headache in jaundice due to catarrhal changes, particularly if the kidneys are not active in the elimination of toxic substances.

Similar symptoms to those just described may occur in cases suffering from paroxysmal hæmoglobinuria, for in this state severe headache, nausea, vomiting, and persistent yawning are often present, with an icteroid discoloration of the skin. The reddish urine, pain in the liver, and sometimes an urticarial eruption will aid the diagnosis of this primary hæmoglobinuria.

In other cases in which no jaundice is present violent headaches, which utterly incapacitate the patient, come on from autointoxication. Thus a man apparently perfectly well goes to bed on a certain night and wakes in the morning feeling a little more drowsy than usual. On rising he may feel a little stupid, and perhaps be slightly vertiginous, but is able to eat his breakfast as heartily as usual. In the course of a few hours the mental heaviness becomes more marked and a pain in the brow develops, which gradually gets worse and worse till it is unbearable. The ordinary remedies for neuralgic headache are futile, and he finds no relief until by the use of a purgative he removes the source of his intoxication, and his kidneys have time to eliminate the toxins already absorbed. Sometimes vomiting comes to his relief, and the emptying of the stomach, produced by the efforts of vomiting, so stimulates his liver and intestines that the process of autointoxication ceases. Some of the intestinal poisons have been isolated by Brieger, Harnack, and others, and have a physiological action like many well-known drugs. Thus one produces effects like those of digitalis, another like those of belladonna, and a third like those of aconite. Pulsating pain and a slow, full pulse may indicate the absorption of the digitalis-like toxin; a flushed face and hot, dry skin, the belladonna-like toxin; and pallor, faintness, and a feeble pulse, if no nausea is present, the presence of the aconite-like toxin. Persons suffering from headache of this type are nearly always much freer from discomfort in the head after such attacks than they have been for some time before.

Brunton has also pointed out that digestive headaches are often associated with an objective and subjective sensation of increased intraocular tension and tenderness on the upper surface of the eyeball, and the author has frequently confirmed this observation.

The headache of *eye-strain* is usually due to insufficiency of the ocular muscles. Most commonly, according to Noyes, the externi (abductors) are the muscles which are the seat of the difficulty. Such headaches may be felt in any part of the head, but are most commonly said to be in the occipital region. If, in association with such headache, immediately after or long after reading, there is blurred vision, pain in the muscles of the eye on suddenly moving the eyeball, any tendency to congestion of the lids or hyperæmia in the conjunctiva over the insertion of the muscle, the diagnosis of headache from eye-strain is practically certain. (See chapter on Eye.) Violent pain in the head may also be due to irritable retina and to astigmatism and spasm of the ciliary muscle. Acute inflammatory processes in any part of the eye may produce severe headache, particularly iritis, the pain of which is very apt to be worse at night.

Whether insufficiency of the ocular muscles or eye-strain can cause "sick headache" by reflex irritation is still undecided, but those ophthalmologists who are inclined to carry the theory of refractive errors in the production of morbid symptoms to excess believe that it can.

Violent headache is often produced by *acute or chronic glaucoma*, and is usually felt about the eyes or orbit. Often it is of a unilateral character, and the sharp, shooting pain causes a false diagnosis of neuralgia to be made, or in some cases the patient is thought to be suffering from migraine, because in addition to unilateral pain there are often nausea, vomiting, and pallor of the face. The examination of the eye will show glaucoma to be present. Quite similar symptoms may appear as the result of a foreign body lodged in the cornea.

The headache associated with *nervous exhaustion* or neurasthenia may be superficial or deep; that is to say, neuralgic or apparently within the skull. It is often associated with some dizziness and vertigo, and is nearly always occipital in character, more rarely appearing over the brows. In addition to the pain, which is generally not very severe, there is often a sense of constriction about the head. Such a headache persists as long as a person who is overworked persists in fatiguing himself, and rapidly disappears when rest is taken. More rarely the pain in the head in neurasthenia is that of migraine, and is complicated by hemianopsia and hemicrania, often by a dilated pupil on the affected side, and flushing and pallor of one side of the face.

Headaches due to *rheumatism* are often quite severe, and are associated with much tenderness of the scalp or muscles covering the

skull. Similar headaches, but more dull in character, are also seen in persons suffering from phosphaturia, and are relieved by benzoate of ammonium.

A headache is a symptom very commonly seen in persons who are subject to the chloral-habit, and it may be general or limited to the forehead. It is commonly associated with vertigo, flushing of the face, and intense heaviness and drowsiness.

Headache of a violent, bursting character may be produced by full doses of nitroglycerin, the salicylates, and quinine, and by the use of large quantities of tobacco.

Leaving the headaches due to functional disturbances not associated with organic change, we pass to those due to organic disease. Headaches due to *renal disease* are of two classes, namely, they are an evidence of uræmia, or they are congestive and due to the high arterial tension so often seen as the result of chronic contracted kidney with its associated conditions of cardiac hypertrophy. Uræmic headache, as pointed out in the chapter on Vomiting, is often associated with nausea or vomiting of a persistent type, and sometimes with diarrhœa, for purging is an effort at elimination. The pain is not of the shooting, darting, or neuralgic type, but dull even if severe, and is often associated with a sensation of fulness in the head. Sometimes the tendency to drowsiness is very marked, and, even if the patient does not sleep, he may seem on the verge of sleep all the time.

These uræmic headaches may occur in any form of renal disease, acute or chronic, which results in uræmia; but, if the cause be chronic contracted kidney, there will be a high arterial pressure, and often a strongly beating heart with an accentuated second sound. This form, with high arterial pressure, will often be relieved by full doses of nitroglycerin, which not only relieves the tension, but also produces an increased renal activity. The urinary examination is of the utmost importance, and no surely correct diagnosis can be made in any case of suspected kidney trouble till this secretion has been examined and found abnormal. (See chapter on Urine.)

While headache is far less common as a symptom of *diabetes* than of nephritis, it occurs in the former disease either as a dull pain with lassitude and depression of spirits or as violent neuralgia.

Headache which is constant, although it usually varies in degree, may be due to *brain-tumor*, and is one of the most important symptoms to be noted in the diagnosis of a case in which such a lesion is suspected. The pain is often worse at night, and is usually more

severe in persons suffering from tumor of the cerebellum than in cases in which the growth is in the cerebrum, probably because cerebellar growths often cause effusion which produces pressure inside the skull. A tumor of the cerebral cortex, as a rule, produces more pain than one in the white matter deeper down. Meningeal growths are also apt to produce severe headache, but bony tumors of the skull often press upon the brain to an extraordinary degree without causing any symptoms.

Headaches due to brain-tumor often have exacerbations with a regularity suggesting malarial disease, and, conversely, care should be taken not to mistake malarial headache for brain-tumor.

After constant headache, the most valuable confirmatory evidence of brain-tumor is papillitis of the optic nerve, which is present in about 80 per cent. of the cases. There may also be vomiting, and convulsions if the growth be in the motor cortex. Local paralysis, indicating the position of the growth, may be entirely absent, or it may exist and yet utterly mislead the physician as to the focal area which is diseased, since cases are on record in which, for example, a hemiplegia has existed and at the post-mortem examination the growth has been found in the frontal lobes. Tumors of the base of the brain cause focal symptoms most commonly, and in addition to unilateral choked disk we find in many such cases ptosis from paralysis of the oculomotor nerve, disturbances in the functions of the trifacial nerve in its sensory filaments, so that painful tic (see chapter on Face and Head) or anæsthesia of the face may be present and complete facial (Bell's) palsy may occur. If the hypoglossal nerve is affected by the pressure, the tongue is protruded to one side, it develops hemiatrophy, and disorders of speech result. Hirt points out that a tumor in the anterior fossa is apt to produce paralysis of the olfactory and oculomotor nerves and the upper branch of the trifacial. A tumor in the pituitary body causes pressure on the chiasm with resulting amaurosis, ptosis from oculomotor palsy, and internal squint from paralysis of the abducens (sixth), anæsthesia of the skin and muscles of the eyebrow, forehead, nose and eye, from involvement of the first division of the trifacial. A tumor of the middle fossa above the dura causes oculomotor palsy (ptosis), patheticus paralysis (downward deviation of the eyeball from paralysis of the superior oblique), amaurosis from pressure on the chiasm. On the other hand, if it is below the dura, the oculomotor, the pathetic, the abducens, and the fifth nerve are paralyzed. When tumors occur in the posterior fossa they cause

paralysis of the trifacial, facial, auditory, glosso-pharyngeal, vagus, spinal accessory, and abducens, or, in other words, cause anæsthesia of the upper part of the face, facial paralysis, deafness, loss of taste, irregular cardiac action, loss of power in sterno-mastoid and trapezius muscles, and internal squint. Tumors of the lenticular and caudate nucleus, the interior portion of the thalamus, the corpus callosum, the fornix, choroid plexus, and of any part of the cerebellum except the vermiform process, may be present without any localizing signs.

Still more localizing symptoms are early paralysis of the oculo-motor nerve from a lesion in the crus, hemianopsia in tumor of the occipital lobe, and tonic convulsions with preservation of consciousness and a staggering gait in tumor of the vermis of the cerebellum.

Should amaurosis be present, very valuable data as to the position of the growth is to be had from a study of the functions of the eye. If the pupils react properly to light, this shows that the optic nerve and tract are intact, or, in other words, that the ocular reflex arc is perfect, and that the lesion must be in the ocular centres further back. On the other hand, if the reflex is absent, the growth is probably in the nerve or tract. (See chapter on the Eye.)

The failure of a pupillary reaction may, however, depend upon amaurosis from lateral hemianopsia, in which case we examine the patient for what is known as "Wernicke's sign of hemiopic pupillary inaction." This is done by throwing the light by the ophthalmoscope so that it falls upon the blind half of the retina. If the pupil does not react, we have in all probability a lesion of the optic tract of that side; whereas, if the pupil does react, we have evidence that the tract is intact and there must be a bilateral lesion of the optic radiations of the occipital lobes, or in the centre of vision in the cortex. (See chapter on Eye.)

Other general symptoms of brain-tumor are slow breathing, particularly when the patient sleeps; a slow pulse, and, as the growth increases, symptoms of cerebral compression.

Care should be taken in cases of constant severe headache that it be not thought due to brain-tumor until the possibility of its being due to syphilitic arteritis is excluded, for the same mental depression, sharp exacerbations of pain, and an increase of pain at night, also occur in this state. There is often nocturnal delirium in these cases. In arteritis due to syphilis the pain is usually diffuse, whereas in tumor or syphilitic gumma it may be localized. Optic neuritis, if present, makes the diagnosis of tumor certain. The chief symptoms

of syphilitic arteritis are headache, giddiness, weakness of groups of muscles, difficulty of speech so that words are dropped out, and very commonly symptoms of general paresis develop. It is, however, often impossible to separate headache due to cerebral syphilis from that due to chronic meningitis or cerebral tumor due to other causes, except by the history of specific infection or manifestations of this disorder in scars or other external signs of syphilis.

Violent headache is the most marked symptom of brain-abscess; but focal symptoms—that is, localized palsy pointing to the area of the abscess—are very often absent, although the localizing symptoms which have just been described as due to tumor may, of course, be due to abscess if it is so placed as to press on nerve-tracts or centres.

The rises of temperature which frequently occur in cerebral abscess are also indicative of the presence of pus, while the more rapid course of the disease, often only one or two weeks, points to abscess rather than tumor. Further than this, choked disk is rare in abscess and common in cases of tumor.

The difficulty of separating the headache of brain-tumor from that due to brain-abscess is very great, for the symptoms with the headache are almost if not quite identical in both cases. One of the most important of the differential points is the history of an injury to the head or of the presence of an infecting focus, which could have caused cerebral abscess.

In some cases of acute cerebral abscess, particularly in children, there is a curious tendency to bore the head into the pillow, or, if the child is still about, the head is rubbed or butted into the wall or against the body of the nurse. These symptoms are, however, absent in the slow, insidious forms.

When the physician has made a diagnosis of cerebral abscess from the headache and associated symptoms, he must not be misled into a reversal of his diagnosis by marked improvement in the patient, who may so far recover as to go back to his occupation, for it sometimes happens that a remission or latent period develops in the subacute forms of abscess. During this apparent remission, however, the temperature is rarely constantly normal, and the patient is anything but well, and chilly sensations may be present.

Severe headache well diffused over the skull, coming on rather rapidly and associated with fever, stiffness of the back of the neck, vomiting, photophobia, delirium, and, finally, stupor and paralysis,

is probably due to meningitis or to tubercular meningitis, effusion at the base of the brain, or, more rarely, to the onset of a severe attack of one of the acute infectious diseases.

If the disease be tubercular meningitis, the head-pains will often be paroxysmal in character, so that the patient will at intervals of varying length give vent to sharp cries, evidently due to a sudden dart of pain. Vomiting may also be present and ocular symptoms develop, such as ptosis, strabismus, and unequal pupils, with a sluggish reaction. The febrile movement will be irregular, now high,

FIG. 185

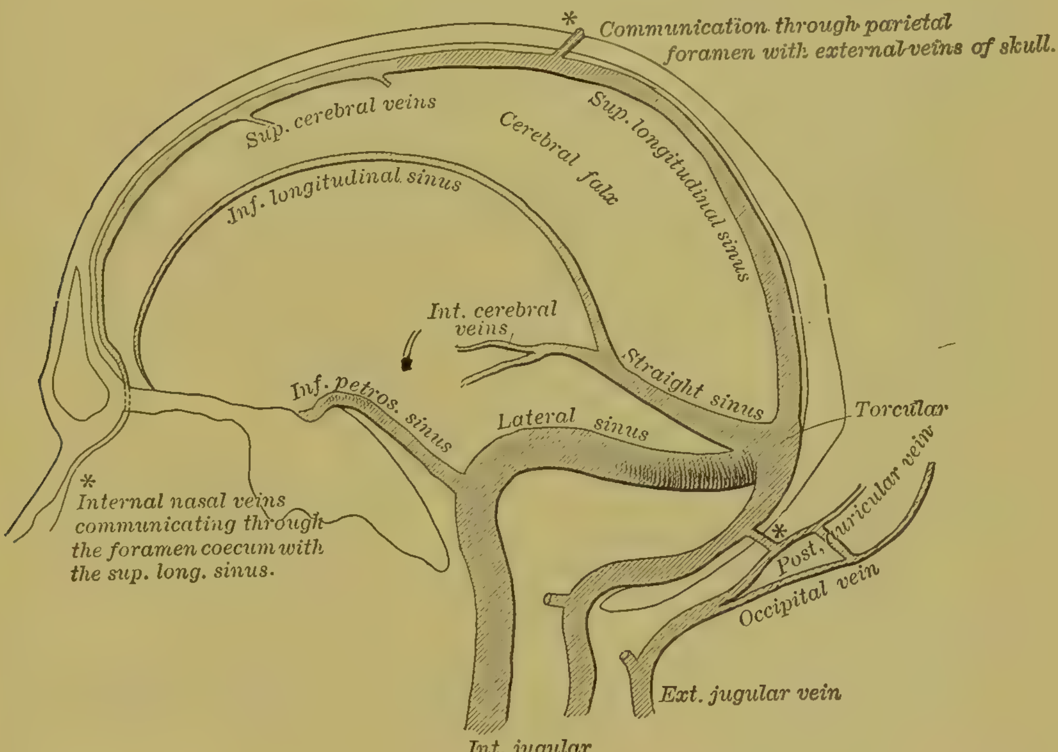


Diagram showing the communications existing between the superior longitudinal and lateral sinuses and the external veins, indicated in the figure by *. (LEUBE.)

then very low; the temper peevish, if consciousness is present; and the skin pale and transparent. In the severe and rapid cases of tubercular meningitis marked delirium comes on, the patient picks the bedclothes, and there are tenderness and stiffness of the nape of the neck. Pulmonary signs of tuberculous disease are often present. Care must be taken that the case is not mistaken for and thought to be typhoid fever, which it may closely resemble in its early stages when headache, malaise, languor, and remitting delirium are present.

The symptoms of meningitis are, however, closely followed by those due to thrombosis of the cerebral sinuses, so closely, indeed, that only the presence of the typical signs of such occlusions can determine the diagnosis. Thus, if the superior longitudinal sinus is affected by thrombosis, there may be epistaxis from distention of the nasal veins, and the temporal veins will be swollen and the near-by tissues œdematous through their close connections with the sinus through the emissary veins of Santorini, which escape from the skull by way of the parietal foramina. (Fig. 185.) In children there is usually in such cases bulging of the fontanelles and heaviness. Somnolence or delirium may be present with many of the characteristic symptoms of meningitis. This condition usually arises in connection with chronic exhausting diseases, such as long-continued diarrhœa and the continued fevers.

Thrombosis of the cavernous sinus is usually accompanied by quite typical symptoms. There is œdema of the eyelids and finally of the entire side of the face on the side of the affected sinus, but this facial symptom may be absent or very fleeting in its duration. Sometimes there is exophthalmia, and if the thrombus is septic a phlegmonous inflammation of the orbital connective tissue may occur. These symptoms are due to the communication between the sinus and the ophthalmic veins. Finally, as pointed out in the chapter on the Face and Head and on the Eye, paralysis of the oculomotor nerve, the ophthalmic branch of the fifth nerve and of the adducens and patheticus may occur, as these nerves pass through the cavernous sinus or in its walls. Nearly always thrombosis of the cavernous sinus results from some diseased processes near-by, as in disease of the middle ear and mastoid. Sometimes the affection is bilateral.

If the lateral sinus is affected by thrombosis, there is usually marked œdema back of the ear, owing to the clot extending to the small veins of the scalp, which pass through the mastoid and posterior condyloid foramina. The external jugular vein on the affected side is partly collapsed particularly on full inspiration (Gerhardt's symptom). Rarely this vein may be unduly distended (Fig. 186). Thrombosis of the lateral sinus occurs far more frequently than that of the other sinuses. Suppurative otitis is its most common cause, and agonizing earache is therefore a symptom often associated with it.

Not only may cerebral thrombosis present symptoms resembling those of meningitis, but in addition those of cerebral abscess.

Violent headache with vertigo, staggering, and confusion of thought,

followed by unconsciousness, may follow meningeal hemorrhage due to disease of the bloodvessels, which are ruptured by some strain or by increased blood-pressure under the influence of stimulants. Hemiplegia or localized spasms may be present. The patient may survive several days in severe cases, or may recover if the hemorrhage is small; but usually a hemorrhage large enough to cause marked symptoms is large enough to cause death.

FIG. 186.

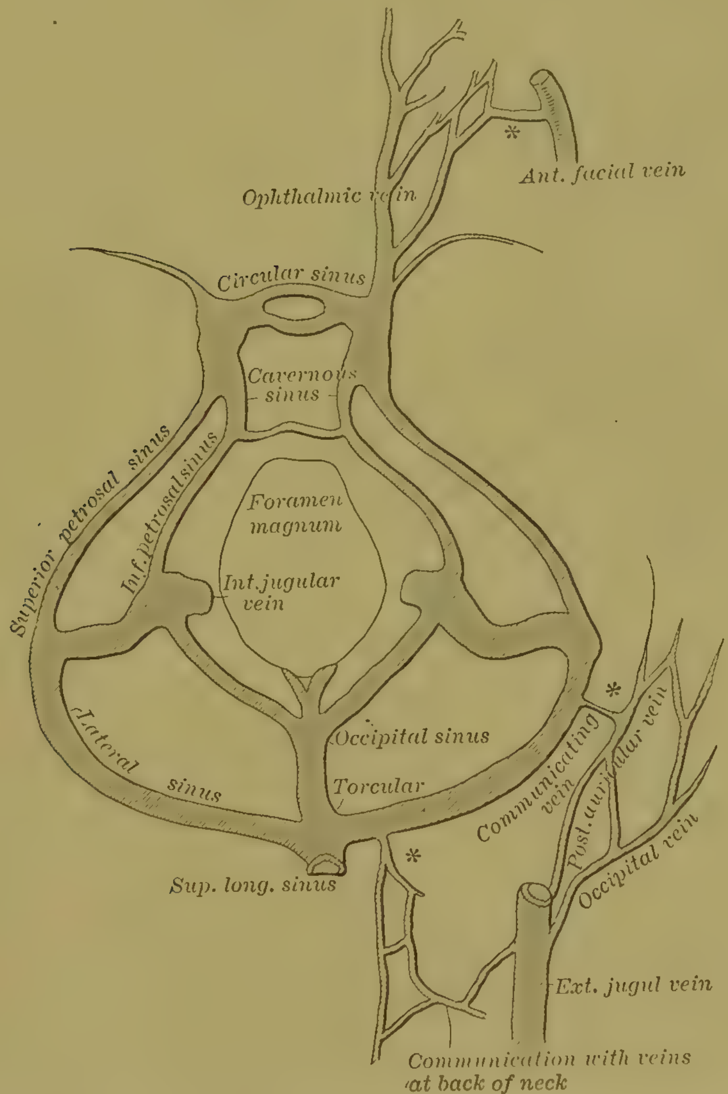


Diagram showing the communication existing between the lateral and cavernous sinuses and the external veins, indicated in the figure by *. (LEUBE.)

The individual affected by a meningeal hemorrhage will usually be plethoric, and, with the symptoms just described, will suffer from photophobia, extreme sensitiveness to the slightest noise, and pain

radiating down the neck and trunk, which occurs in paroxysms. Localized paralysis is rarely present.

The presence of severe vertical headache in a middle-aged person who is insane and who is a male may indicate pachymeningitis interna hemorrhagica (hæmatoma of the dura); but usually the insane patient does not complain, and an ante-mortem diagnosis of this state is not made.

Headache resulting from heat-stroke or thermic fever is usually the result of meningeal congestion or inflammation, and is one of the most annoying symptoms of convalescence. It is apt to be greatly increased by moving the head, and is relieved by venesection.

The earlier stages of smallpox and pneumonia of the croupous type are often periods of violent headache, which symptom in the former instance decreases with the appearance of the rash, and in the case of croupous pneumonia so closely resembles the headache and associated symptoms of meningitis that a diagnosis in the absence of pulmonary signs may be impossible. In every case in which such symptoms occur the lungs should be examined.

Headache is a constant symptom in many cases of typhoid fever in the early stages, but the peculiar tongue (see chapter on Tongue), the tendency to diarrhœa, the general systemic symptoms, and the facies of the patient will usually make its cause clear. More or less violent headache is often seen in measles, and depends probably to a great extent upon the engorgement of the nasal mucous membrane, or, in other words, has the same causative factor as has an acute "cold in the head" in producing cephalalgia.

Gruening is quoted by de Schweinitz as asserting that early morning headache is often due to nasal catarrh. This is, of course, only true if digestive troubles, which are often due to alcohol, and renal disorders are excluded. Severe morning headache, or dull headache on just waking up may be due to nocturnal attacks of epilepsy, of which the patient is ignorant. If the tongue is bitten or the bed wet with urine, this diagnosis is strongly indorsed.

Violent headache is often present during the febrile stage of intermittent fever and is often a complicating symptom of fever of the remittent type. In this connection the physician should remember that violent neuralgia of the supraorbital nerve is sometimes due to malarial poisoning, and is called "brow ague."

Headache is often due to anæmia, whether it be the result of hemorrhage or of the deficient formation of blood. The pain is usually

frontal, there are often giddiness on movement, palpitation of the heart, a peculiar sensation in the head, and pallor of the skin. An examination of the blood will usually reveal the cause to be in this tissue.

Headache sometimes results from valvular heart disease. Thus in mitral regurgitation it is often associated with vertigo, stupor, sleepiness, and, as night approaches, a mild delirium may come on. Its probable cause is congestion of the brain.

Rarely intracranial aneurisms produce headache, and when they are of the diffuse miliary variety this symptom may be a prodromal one before an attack of apoplexy. Large aneurisms may, however, exist without severe headache, and the position of the pain in no way indicates the seat of the aneurism, save that aneurism of the basilar artery may cause occipital pain. Auscultation might possibly reveal a murmur.

Headache may also arise from disease of the skull bones, either caries, ostitis, or periostitis, which result from injury, infection by syphilis or other infecting cause, such as typhoid fever or tuberculosis; but there is nothing diagnostic about the headache in these cases save that it is generally most severe in the area involved, and pressure over that part may elicit more or less pain or tenderness.

Violent neuralgia or shooting headache may be produced by exposure to cold, with resulting inflammation of the nerve-sheath; by dental caries, and by middle-ear disease or disease in the external auditory canal.

When headache is present in the course of croupous pneumonia it often lasts till crisis, but in some cases ceases by the third day. The chest should always be carefully examined in all cases of severe headache with fever for signs of pulmonary disease.

Vertigo.

Vertigo is a condition in which the patient feels as if he were losing his equilibrium. Sometimes he feels as he were whirling around from right to left or left to right, sometimes as if falling forward or backward, and sometimes he seems stationary, while all his surroundings whirl round or rise up to or fall away from him. Although a symptom which in itself lacks danger, vertigo always produces great discomfort, if not fear. Vertigo arises from the patient being subjected to a whirling motion, from rough sea-voyages, and from indigestion, deficient circulation, or excessive cerebral

congestion. Often it is due to the cerebral anæmia arising from excessive hemorrhage. When it arises from indigestion it is probably due to reflex irritation, and perhaps to the absorption of toxic materials.

Vertigo as a symptom has a far more serious significance when it arises from organic disease. The most common lesions which cause it are middle-ear disease, Ménière's disease, tumors of the cerebellum, of the pons, of the crura cerebri, and the corpora quadrigemina. Vertigo also is not only a premonitory sign of an epileptic attack, but in the eplieptic state called *petit mal* or minor epilepsy it is often the only symptom. In persons with atheromatous arteries it is very common, and sometimes it is a persistent symptom for some days before an apoplectic seizure. It is also present in disseminated sclerosis. Finally, many drugs, such as quinine and the salicylates, may produce it.

As the diagnostic points connected with most of the lesions here named are discussed elsewhere in this book, only Ménière's disease will be mentioned at this place. In addition to vertigo the characteristic symptoms of Ménière's disease are vomiting, noises in the ears, and, finally, deafness. The vertigo may be so severe that the patient falls to the ground. Aural examinations are usually futile in discovering any cause. Some authorities believe the disease to be due to a neurosis of the vasomotor nerves supplying the semicircular canals.

An unknown form of vertigo in America, the paralyzing vertigo of Switzerland, described by Gerlic, is a paroxysmal vertigo with great loss of power in the limbs, partial ptosis, and preserved consciousness.

CHAPTER III.

COMA OR UNCONSCIOUSNESS.

COMA is a condition of unconsciousness or insensibility from which the patient can be roused but partially or not at all, and it may arise from injuries to the head, while the patient is in otherwise perfect health, which injuries produce laceration of the brain-substance, cerebral or meningeal hemorrhage, or concussion. Again, it may be due to the influence of certain poisons, as alcohol, opium, chloral, cannabis indica, very large amounts of the bromides, or poisonous doses of other narcotics. Thirdly, it may arise from auto-intoxication, as in uræmia resulting from renal disease; in cases of profound exhausting disease, like typhoid fever or ulcerative endocarditis; in cases of diabetes, or from acute yellow atrophy of the liver and pernicious malarial fever. Fourth, as a coincident symptom or sequel of hemorrhage into the brain (apoplexy), as the result of an epileptic attack, of a cerebral embolism or thrombosis, of thrombosis of the cerebral sinuses, of cerebral abscess, of pachymeningitis, leptomeningitis, or cerebro-spinal meningitis, of cerebral syphilis, of general paralysis, multiple sclerosis, and heat-stroke. The various points in connection with the diagnosis of coma from head-injuries are to be found in surgical treatises, and the history of a head-injury or the very presence of any injuries to the head is an important point to be sought after in the diagnosis. Care should be taken, however, that any head-injuries found to be present are not the result of a fall due to the onset of sudden unconsciousness, rather than that they are the cause of the coma.

The coma of *acute alcoholic poisoning* is characterized by profound insensibility, great muscular relaxation, loss of the ocular reflexes, and great fulness of the bloodvessels of the neck and face in the early stages, but finally ghastly pallor of the face as the coma deepens on the approach of death. The skin is moist and warm at first, but afterward becomes cold. The pupils are usually moderately dilated; the pulse is rapid, at first strong, then more and more feeble; and the respiration stertorous and heavy. The sphincters, as a rule, are not relaxed, although they may be so in rare cases.

The bodily temperature in severe alcoholic poisoning progressively falls from 1° to 6° F. below normal.

Alcoholic coma is to be separated from that due to opium-poisoning by the absence of the contracted pupils and slow breathing of the latter condition, in addition to the other symptoms named below in discussing that condition; from coma due to cranial fracture by the absence of any history or sign of head-injury[1]; from chloral-poisoning by the history, the greater fall of body-temperature and the great feebleness of the heart and respiration produced by chloral. It may be impossible to separate alcoholic poisoning from that of cannabis-indica-poisoning except for the fairly strong pulse generally found in the latter condition, and the history of the patient having taken the hemp or complained of the peculiar sense of prolongation of time before the coma came on.

The symptoms accompanying the coma of *opium-poisoning* are heavy sleep, preceding the deep unconsciousness, during which the patient can usually be aroused by shouting in his ear or by violent shaking, but sinks back into slumber at once on being undisturbed. The face is suffused and reddened and may be finally distinctly cyanotic, and the breathing puffing and stertorous. When the patient is awakened he breathes more rapidly, and for this reason the duskiness of the face disappears and the normal hue returns. Death never occurs in the second stage of opium-poisoning from the poison alone; but, if disease is present, death may take place at this time. The pupils are contracted to pin-points. The third or fatal stage emerges from the second by a gradual process, so that no abrupt line of separation can be noted. The face becomes at first more cyanotic, then pale and livid; the respirations, which have been eight to ten in the minute, are now only four or five; and, finally, such prolonged pauses occur that all hope of another respiration is lost by the attendant. While the slow breathing is at first deep, it now rapidly becomes shallow, and relaxation is present to the greatest degree. The skin, previously dry, is wet with the sweat of death; the patient is so deeply narcotized that nothing can arouse him and he dies from respiratory failure, although the heart ceases almost simultaneously from the asphyxia. The pupils do not dilate in the third stage, except in the relaxation of death.

In view of the frequency with which alcoholic and opium-poison-

[1] The physician must not forget that a fall from alcoholism may result in cranial fracture.

ing are confused, the following table is appended, which will be found of value in making a differential diagnosis as to the condition of the patient:

Opium-poisoning and Alcoholism.

Opium-poisoning.	*Alcoholism.*
1. Pupils contracted.	1. Pupils normal or dilated.
2. Respiration and pulse slow and full.	2. Respiration nearly normal; pulse rapid, and finally feeble.
3. Face suffused and cyanosed.	3. Face may be pallid.
4 Skin warmer than in alcoholic poisoning.	4. Skin cool, perhaps moist.
5. Pulse slow, strong, and full till late in poisoning.	5. Pulse rapid, at first strong, then weak.

There is scarcely any difference as to consciousness in these two conditions.

When a *poisonous dose of chloral* is taken by man the person soon falls asleep and then sinks into a deep coma. The respirations become at first slow and labored, then shallow and feeble. The pulse, at first perhaps a little slowed, soon becomes rapid, thready, and shuttle-like, and is finally lost at the wrist. The face is white and livid, the forehead and the hands covered with a cold sweat, and the pupils, which are at first contracted, soon become widely dilated. Absolute muscular relaxation is present, and it is impossible to arouse the patient.

The coma of *uræmia* may come on gradually, but most commonly its onset is sudden and follows a uræmic convulsion. It possesses no diagnostic sign or signs which clearly separate it from the unconsciousness or coma following epileptic attacks, and, as the uræmic convulsion is usually typically epileptic in character, the differential diagnosis is very difficult. An examination of the urine, if it can be obtained by the catheter or otherwise, will indicate, but not prove, the presence of uræmia if albumin be found in either large or small amounts, and the presence of very little urine in the bladder, indicating anuria, may be of some diagnostic significance. On the other hand, if the uræmia be due to chronic contracted kidney, the urine may be plentiful, the albumin scanty, but the low specific gravity be noteworthy. The pulse is usually very slow, often but forty to fifty, but the arterial tension is high, so that the artery feels hard and unyielding. The temperature of the body is usually very low in those severe cases which are free from convulsions and have a progressively downward course; so low a point as 91° to 95° being sometimes reached, at which time the patient is usually moribund from collapse. When convulsions are present the temperature may

rise as high as 108°, and there may be in some cases a severe chill, followed by fever, and this again by collapse. The respiration is nearly always very deep, and sometimes very much quickened. If a preceding history of prolonged nausea, attacks of colliquative diarrhœa, and vertiginous symptoms can be discovered as symptoms leading up to the unconsciousness, these will add to the array of uræmic probabilities. The coma of uræmia is not necessarily a fatal symptom. Even in very severe cases remarkable recoveries sometimes occur.

Coma resulting from *diabetes mellitus* is of far graver import, as it commonly terminates the patient's life. There may or may not be any prodromes, and there may or may not be any history of an exciting cause for the coma to be superimposed upon the already present disorder of nutrition. Sometimes it is provoked by severe exercise or great mental strain or emotion. When unconsciousness does not come on at once the patient, after suffering from nausea, headache, and respiratory oppression, suddenly becomes anxious, delirious and violent, then drowsy and deeply comatose. The pulse is not particularly noteworthy, but is usually full and not very tense. The respirations are deep and often very noisy, but at about the normal rate, although sometimes they may be rapid in the condition called diabetic dyspnœa. The body-temperature falls very greatly, even below 90° F. The respiratory changes and those in temperature may, therefore, be very much like those of uræmia; but in association with the coma of diabetes mellitus there are two pathognomonic symptoms: First, the sweet odor of the breath, which smells like the aroma of a pear or apple, or a faint odor of chloroform; and, second, the presence of sugar in the urine, which secretion becomes dark red on the addition of chloride of iron.

Ordinary coma is rare in *typhoid fever;* it is usually replaced by what is called coma-vigil, in which the patient, in a semi-unconscious state, keeps muttering as half awake, day or night. It is a grave sign.

The coma of *acute yellow atrophy of the liver* is generally preceded by headache, nausea, anorexia, and perhaps fever, followed by nervous excitement or restlessness, and then mental hebetude, which is often accompanied by a noisy delirium which may amount to mania. Finally, after several days, coma comes on and gradually becomes more and more profound till death takes place. Some of these symptoms resemble those of uræmia or diabetic poisoning, but the coma of acute yellow atrophy has in addition these characteristic

signs, namely, jaundice, bile-stained urine, marked shrinking of the liver-dulness, enlargement of the spleen, and hemorrhages into the skin, or these effusions may take place into the bowels and stomach. The urine is singularly free from urea, but contains leucin and tyrosin in large amounts. (See chapter on Urine for description of leucin and tyrosin crystals.)

The coma of *apoplexy* may be sudden or gradual in its onset; generally it rapidly appears after the first symptoms of cerebral hemorrhage develop. The loss of consciousness may be partial or absolute, generally the latter if the leakage from a ruptured vessel be great. The respirations become stertorous, generally more rapid than normal, and, if a fatal result is in prospect, are rhythmically irregular; that is, they are now very slow, then gain in speed gradually till they become very fast, then the speed and vigor gradually fall till they are as feeble and slow as before (Cheyne-Stokes respiration). The history of preceding paralysis in one side, or the presence of this loss of power if it can be demonstrated, the unequal pupils, the drawing of the face away from the paralyzed side, a strong, bounding pulse, and generally raised temperature finish the clinical picture of the coma of cerebral hemorrhage. If death does not ensue, consciousness may return, and the patient progress to recovery; but sometimes after several days of apparent convalescence a secondary fatal irritative coma comes on, associated with high fever. This is usually of ominous portent and is readily recognized because of the history. (See chapters on the Arm and on the Leg and on Hemiplegia.)

The coma of cerebral hemorrhage is unfortunately often taken for acute alcoholism, particularly as the latter state often produces the hemorrhage. The following table is designed to separate them:

Acute Alcoholism and Apoplexy.

Alcoholism.	*Cerebral Hemorrhage.*
1. Pulse rapid, compressible, and weak.	1. Pulse apt to be strong and slow.
2. Skin moist, or relaxed and cool.	2. Skin hot or dry.
3. Bodily temperature lowered.	3. Bodily temperature raised.
4. Pupils equally contracted or dilated; generally dilated.	4. Pupils unequal.
5. No hemiplegia.	5. Hemiplegia; one side tossed, the other remaining motionless.
6. Breathing not so stertorous nor so one-sided in lips.	6. Respiration stertorous, the lips being inflated on one side on expiration.
7. No facial palsy.	7. Facial palsy.
8. Unconsciousness may not be complete.	8. Unconsciousness complete.

The smell of alcohol in the breath is no guide, as acute alcoholism may have caused the rupture of a cerebral bloodvessel.

Coma due to *cerebral softening*, following embolism or thrombosis, has no signs other than those discussed in the diagnosis of these lesions in connection with hemiplegia (which see). Coma due to thrombosis of the sinuses of the brain is accompanied by the following diagnostic symptoms, namely, irritation or paralysis of the cranial nerves resulting in strabismus, nystagmus, and lockjaw, stiffness of the neck, and clonic spasms. If the cavernous sinus is thrombosed, there will generally be found stasis of the veins in the eye, which means retinal congestion. The eyeball may be protruded, the eyelids swollen, and perhaps loss of function in the oculomotor nerve may be present, causing ptosis, and, if the abducens is affected, causing internal strabismus from paralysis of the external rectus. If the transverse sinus is involved, there will probably be œdema behind the ear, and, if the petrosal sinus or internal jugular be obstructed, the proximal part of the vein collapses. Thrombosis of the superior longitudinal sinus causes epistaxis and engorgement of the temporal veins. Thrombosis of any of these sinuses, however, may be present without these signs. Coma due to subdural hemorrhage (pachymeningitis interna hemorrhagica) is peculiar in the fact that its onset is usually very slow, and the signs of nervous irritation last a long time and are quite violent, often amounting to epileptic paroxysms. Commonly, too, there will be rigidity of one limb, but the cranial nerves usually escape. The coma usually follows these signs, and the condition is peculiarly common in the chronic insane and in paretic dements.

Coma from *cerebral abscess* is accompanied by symptoms closely resembling those of acute meningitis. The patient is dull and delirious; has headache, fever, and often has a hyperpyrexia. The sensibility becomes less and less, and deepens into the coma which ends in death if relief is not given. The localizing symptoms of paralysis may indicate that a lesion is in a certain part of the brain; but generally these signs are absent, because cerebral abscess is usually in the frontal lobes. If there is a history of injury, purulent otitis, infectious disease involving other parts, such as septicæmia from wounds or empyema, and if there are vertigo, vomiting and headache, fever, and an absence of choked disk of the optic nerve, the diagnosis is probably cerebral abscess; but a long duration of months is no sign that it is not abscess, as these cases often run a very prolonged course.

The coma of *purulent lepto-meningitis* resembles that of abscess in many of its associated symptoms, but the intense headache, the rapid development of delirium and unconsciousness, the stiffness of the neck, the optic neuritis and disturbed movements of the ocular muscles, combined with the absence of a history of septic absorption, may make a differential diagnosis possible. Purulent lepto-meningitis is rare, but it sometimes occurs in association with croupous pneumonia, and the presence of this disease will point to the cause of the coma. The coma due to *epidemic cerebro-spinal meningitis* is diagnosed by the characteristic rigidity of the neck, excessive headache preceding the unconsciousness, the disturbances of the cranial nerves producing strabismus, unilateral or bilateral ptosis, nystagmus, impaired pupillary reaction, mydriasis and myosis. The face is often painfully distorted. The presence of an epidemic, of course, makes the diagnosis clear.

Cerebral syphilis may result in the production of coma by producing hemorrhage, embolism, arteritis, tumor of the brain, or almost any other lesion, and its diagnosis as the cause of an attack of coma is not easy. Of course, a history of syphilitic infection and the presence of symptoms of this condition in a patient who is too young to have secondary arterial changes from age render the probability of syphilis as a cause very great. Scars on the skin (see chapter on Skin) may show specific taint.

When coma results from *general paralysis* it usually succeeds the peculiar epileptic attacks which come on late in that disease, and the history of delusions, tremor of the hands, peculiar speech, loss of the reflexes, with earlier milder attacks, like the one before us, combined with the age of the patient, render a diagnosis possible.

Practically identical symptoms may attend the development of coma from *multiple sclerosis*, and without the history of the latter affection the diagnosis may be impossible. If this history shows a spastic gait and intention-tremor, nystagmus, mental weakness, and heightened reflexes, the probability of the attack being due to multiple sclerosis is increased.

Coma is sometimes seen as a later manifestation of *Addison's disease*, and it often develops very suddenly.

Heat-stroke produces coma as one of its almost constant symptoms. The history of exposure to heat and the hyperpyrexia are the two diagnostic points of importance. (See Fever.)

Sudden unconsciousness may arise from *heart-failure* due to disease or fright; we call this fainting. Frequent attacks of this character should cause the physician to listen to the heart to discover if there is valvular disease, particularly aortic stenosis and fatty heart, and he should be on the outlook for renal difficulty. Sometimes sudden unconsciousness will be due to *petit mal* or minor epilepsy.

CHAPTER IV.[1]

CONVULSIONS OR GENERAL SPASMS.

The convulsions of epilepsy in its various forms—Of infancy—Of hysteria—Tetanic convulsions—Spasms—Chorea.

A CONVULSION is a condition in which by reason of sudden tonic or clonic contractions of groups of muscles the body in whole or in part is thrown into spasmodic movements. Convulsions can be divided into those which are clonic or epileptiform and those which are tonic or tetanic. Further, it is a general rule that convulsions which are epileptiform or clonic in character have their origin in the cerebral cortex, while those of tetanic or rigid type arise from excitation of the motor tracts in the spinal cord. The clonic variety of convulsions are represented by idiopathic, traumatic, reflex, and syphilitic epilepsy, hysterical convulsions of an epileptic type, uræmic convulsions, and those convulsions which arise from the presence of growths or other sources of irritation in the cerebral cortex. Certain poisons may also rarely produce such attacks, notably lead and alcohol, and sometimes malingerers imitate very successfully the epileptic paroxysm.

The convulsion in *epilepsy* is characterized in some cases by the primary appearance of an aura—that is, a sensation in some part of the body, which the patient discovers comes on before each convulsion. This aura may be of any character and appear in any part. Most commonly it is sensory, and is as if a cloud or wave was passing up the body to the head. As the sensation reaches the head, the patient may utter the peculiar epileptic cry or sigh, and with this sound the patient becomes rigid from momentary tonic spasm of the muscles. This spasm now relaxes for an instant, and then the patient's muscles pass into a state of alternate relaxation and contraction which throws the patient's body from one place to another.

The primary tonic spasm of the face produces risus sardonicus in some cases; the head is often drawn to one side, the eyes commonly turned to the same side, and the lower jaw locked tightly against the upper jaw. The arms are strongly flexed at the elbows, the hands

[1] For local spasms, see chapters dealing with face and head, hand and arm, and feet and legs.

flexed at the wrist, and the fingers bent into the palm of the hand with great force. As a rule, the evidences of the powerful flexors overcoming the extensor muscles predominate; but sometimes the reverse is the case, and forcible, rigid extension of the parts affected takes place. The duration of these tonic contractions rarely exceeds two minutes, and in most cases is limited to but a few seconds.

It is followed by the clonic spasms, already described, which are ushered in by more or less violent tossings, but whose onset is forewarned by peculiar vibratory thrills, which run through all the affected muscles. The eyelids tremble, the body changes its position ever so slightly, and then, as if the vibrations gained greater and greater power with each moment, the fibrillary tremors give way to muscular contraction. The expression of the face, which in the preceding stage was set and firm, is now constantly changed by the movements of the facial muscles; the jaws, no longer locked together, are gnashed and crunched one upon the other; the tongue is alternately protruded and drawn back, and, as a consequence, is often caught between the teeth and lacerated. The excessive movements of the muscles of mastication force the increased quantities of liquid secreted by the salivary glands from the mouth in the form of froth, which is often stained with blood by reason of the injuries to the tongue. The constancy of the convulsive movements now becomes less and less marked; well-developed remissions occur between each toss of the body, until the movements cease entirely; but it should be constantly borne in mind that the prolongation of the remissions does not produce any decrease in the severity of the intervening spasm, the final spasm often being even more violent than the first.

The intense discoloration of the face begins to pass away as soon as the remissions, by their length, permit the blood to be oxygenated, its disappearance being temporarily arrested by each paroxysm. Finally, the spasms having ceased, the patient lies before us relaxed, unconscious, and exhausted, and passes into a deep sleep or coma, which lasts a variable length of time, and from which he cannot be aroused, except very rarely, and then with great difficulty.

When one part of the body is involved in an epileptic paroxysm, the rest of it escaping, the condition is one of *Jacksonian epilepsy*. By far the most important of its peculiar signs is the character of the onset, which always begins, in the typical Jacksonian form, in some peripheral portion of the body, and most frequently in the muscles of the thumb or hand, so that for the moment the convul-

sive movements are localized. They may remain localized at the point of origin, or immediately diffuse themselves over muscle after muscle until all the arm, leg, or other groups of muscles are involved. It is of the greatest importance, however, that the reader should keep the aura of an attack separated in his mind from the onset, remembering that the term onset is here used by the writer to designate the beginning of the period following the aura, if there be one. Jacksonian epilepsy may be of almost any severity. In rare cases only one muscle may suffer throughout an entire attack, but in others the entire body may be at last convulsed. There may or may not be loss of consciousness, its presence or absence being dependent upon the seat of the lesion in the brain and the severity of the attack. In those instances in which only a few localized muscles are involved consciousness is more commonly preserved than lost.

Typical Jacksonian epilepsy may develop in the course of general paresis.

Epileptiform convulsions may be divided into two classes, in one of which the patient suffers from a single convulsion, the result of a cerebral hemorrhage, and in the other the changes produced by the hemorrhage result in epileptic attacks. When the convulsion occurs at the time of an apoplectic effusion it is generally Jacksonian in character ; that is to say, one muscle or a group of muscles is involved, or, if not this, the attack is, at most, only unilateral. Further than this, it is always associated with the symptoms of apoplexy as generally seen, for there are inequality of the pupils, drawing of the face to one side, and a consequent hemiplegia which lasts indefinitely. Of the attack itself it may be said that, so far as the movements are concerned, they differ in no way from those of the true epileptic seizure; but it should be remembered that hemiplegia often follows ordinary idiopathic epilepsies ; so that the fact that hemiplegia is permanent, and is not temporary, is more of a sign that the attack is due to hemorrhage than the actual paralysis is. It should also be remembered that apoplexy may complicate epilepsy, being produced by the convulsion. In a considerable number of cases of epilepsy it will be found that the convulsions succeeded an attack of paralysis, which was sudden in onset and possessed the characteristics of vascular rupture. In some persons the history of this attack is very indistinct, owing to its occurrence in early life; while in others the paralysis has been so slight or temporary as not to bear any relation in the mind of the patient with the convulsive

seizures following, which, in many cases, do not occur for some time after. The attack may not leave a trace of loss of power behind it, but the convulsions continue, and closely resemble the so-called idiopathic form of the disease. The writer also wishes to call attention to the fact that the palsy and convulsions are not always due to hemorrhage, but to any pathological cerebral change. Heart disease, by causing embolism, may bring them on, and rheumatism, syphilis, and puerperal sepsis may all produce a softening of the cortex, with an epileptic state following the paralysis.

We can very readily divide post-hemiplegic epilepsy into two classes, for we find that in about one-half of the cases the convulsion occurs along with the paralysis, and then follows at intervals, while in the other half the paralysis is not followed by convulsive seizures for weeks, months, or years.

Post-hemiplegic epilepsy may occur at any age, but there can be no doubt that it far more commonly occurs in children than in adults. In at least two-thirds of the cases the onset is before five years of age, and in nearly one-half it is during the first two years of life. Very curious results are reached if the statistics of the affection are analyzed—results which are quite unexplainable unless by hypothesis. Indeed, they tend to overturn many of our preconceived ideas. Thus, it will be found that in the cases which date from infancy females are twice as numerous as males, but in cases after five years of age there is no difference between the frequency in the two sexes. One of the theories of these infantile cases has been that they were produced by the use of instruments during labor, and repeated post-mortem examinations have confirmed the possibility of this occurrence. On the other hand, every obstetrician knows that the birth of a boy generally means a more difficult labor than that of a girl, owing to the greater size of the head in the male child. *A priori* reasoning would seem to show, therefore, that the heads of male children would, accordingly, have instruments applied most frequently, and consequently that infantile cerebral trouble would be the result more commonly in males than in females ; but, as has been said, this conclusion is contradicted by the facts. Another symptom of great interest is that the paralysis in the infantile cases is more frequently on the left side than the right, but after the fifth year it is equally common on both sides.

The writer has already spoken of the fact that the convulsions may occur along with the first attack of paralysis, and continue, or that

an interval may occur between the attack and the subsequent paroxysm. The chronic recurrent fits date from the onset in about one-third of the cases, but it is not uncommon for the paralysis to occur in infancy and the epilepsy to begin at puberty. It would seem that cells injured in early life may lay undisturbed till the increased demands of maturity call them out into diseased action. This prolonged interval occurring so commonly in children separates them from adults in this disease, for in the latter class it is very rare for the epilepsy to be delayed for more than one year.

A distinct aura is present in about five-sixths of the cases, and is consequently far more frequent than in the ordinary idiopathic disorder. When the reader considers the etiology of this affection it will be clear to him that these conditions are virtually forms of Jacksonian epilepsy so called, at least so far as the causative lesions are concerned.

The frequency with which post-hemiplegic epilepsy comes on in the hemiplegia of childhood has been very recently studied, and the conclusion reached that its occurrence is quite common. Thus in Osler's cases twenty children out of ninety-seven suffered from it. In the eighty cases collected by Gaudard eleven children had hemiplegic epilepsy, and sixty-six children out of one hundred cases collected by Wallenburg were epileptic after hemiplegia. In another series of cases collected by Osler fifteen children out of twenty-three were thus affected.

Syphilitic epilepsy is only one of the many nervous affections which afflict those who may be so unfortunate as to contract this disease. There can be no doubt that syphilis produces an enormous amount of epilepsy, and the presence of epilepsy in a person in whom the slightest suspicion of a specific taint exists should cause him to be instantly placed under antisyphilitic treatment. That this is true is evidenced by the statements of the foremost neurologists the world has ever known, for we find no less noted a writer than Charcot stating that epilepsy is the most frequent manifestation of cerebral syphilis, and the equally eminent syphilographer, Fournier, has insisted most strongly on this point, as have also Bravais and M. Lagneau. In England, Hughlings-Jackson, Broadbet, Todd, and Buzzard have promulgated this doctrine, and in America Weir Mitchell, Spitzka, Wood, and Carter Gray have recorded their belief in it, as have also Nothnagel and many equally eminent Germans. Indeed, it would be difficult to name any one statement in medicine which receives more widespread assent on all sides than does this.

The symptoms of syphilitic epilepsy really differ in no way from those of the simple idiopathic variety, but some points peculiar to this form of the affection are well worthy of attention.

In 118 cases of syphilitic epilepsy Echeverria found the symptoms of headache in forty-five males and thirty-eight females, or 70.30 per cent. of them all.

In fifty-nine patients præcordial pain was found in twenty-seven males and thirty-two females, or 50 per cent. of the whole number of cases.

Of the eighty-three patients with cephalalgia ten males and sixteen females had parietal pain, and eleven males and eight females suffered from pain in the temples, while nine males and seven females suffered from occipital pain. In the remaining twenty-two cases the headache was felt all over the head.

The peculiarity of the cephalalgia of syphilis, when complicated with epilepsy, is the constancy with which it annoys or agonizes the patient, always being present to some extent, and frequently exacerbated toward nightfall or during the night, generally getting worse until the convulsion breaks forth, or it may in some instances abate as the storm approaches. Indeed, many syphilographers believe this to be the rule rather than the exception. There is certainly something very typical about the syphilitic headache which, nevertheless, baffles the descriptions that one would like to give of it. Once seen it can rarely be mistaken for anything else, and even the first view of such a case must impress the careful observer with several salient points. The face, one notices, expresses constant suffering, or at least distress and weariness, and the unrelenting character of the pain seems to crush the patient's vitality and liveliness with an iron heel. If spoken to, the man, who has been resting his head on the hands, will either answer slowly and painfully in monosyllables, or, gradually raising the face to that of the questioner, give an answer, and once more return to his former position. These symptoms are not, of course, pathognomonic, but they are certainly characteristic. The pain, too, is in other ways peculiar, and Charcot has expressed the opinion that the crossed character of the pain in this disease is of value, as it points to the motor zone. Indeed, he regards this headache as typical of the disease, particularly when it is, as it generally is, bilateral; that is, in both temples or both occipital regions at the same time.

In the place of the headache we may have, as prodromal symp-

toms, slight loss of memory, unwonted slowness of speech, general lassitude, and especially a lack of willingness to make mental exertion. Somnolence may be excessive, and, if any of these symptoms are seen in a person whose history is syphilitic, they should be regarded as warnings of an approaching crisis of epilepsy or of some other cerebral disorder. The optic disks should be carefully examined, for in many, but not all, cases evidence of brain disease may be noted in the eye. This is particularly true of syphilitic epilepsy as contrasted with its other forms.

There is also one symptom which may occur early in syphilitic epilepsy, or sometimes only late in the disease, namely, repeated partial, passing palsies, which, while they may be in some cases hysterical, are in the syphilitic almost pathognomonic of brain-involvement—a momentary weakness of one arm ; a slight drawing of the face to one side, which disappears in a few hours ; a temporary dragging of the toe ; a partial aphasia which appears and disappears ; a squint which to-morrow leaves no trace behind it. A symptom which has been asserted as being frequent in this disease is the common occurrence of nocturnal attacks; indeed, cases have been reported by Charcot and Lagneau in which this was the case, but there are similar instances, by the score, in ordinary idiopathic cases.

In syphilitic epilepsy there are often well-marked psychical disturbances with incomplete palsies, which, curiously enough, rarely involve the cranial nerves, as has been particularly noted by Heubner; or there may be an excess of psychical disturbance with a minor epileptic convulsion, and with involvement of the basal cranial nerves.

It is important to determine whether *idiopathic epilepsy* can be separated from that due to *syphilis* simply by the symptoms. Of course, this is a very difficult question to decide ; but the answer to a question of this character ought to be that, so far as the convulsion itself is concerned, it is not possible to separate them. If, however, we can obtain any history, the matter becomes much more simple. It is characteristic of syphilis to have severe darting or aching pains in the tibiæ, particularly at night ; and it is also characteristic of syphilitic epilepsy to have severe frontal headache before the attack, while in idiopathic epilepsy this pain generally follows the seizure.

Fournier, in his lectures on epilepsy, in the Louvain, in Paris, in 1875, gave a summary of his views as follows :

1. In *syphilitic epilepsy* there is nearly always absence of the shrill cry at the onset, so characteristic of the idiopathic variety.

2. There is frequently paralysis immediately after the attacks.
3. The seizure is incomplete or unilateral in character.
4. Attacks constantly increase in severity.

A therapeutic point, which may be used with the greatest success, is the administration of iodide of potassium in large doses. If the epilepsy be syphilitic, it will rapidly become less severe, and enormous amounts of the drug will be borne with impunity. As much as 450 grains in twenty-four hours will often do good.

It has been thought by some that the mental hebetude between the attacks is greater in syphilitics than in others. This depends very largely on the area of the cerebrum involved, and not upon the disease itself.

Of course, if there is a history of a chancre or any syphilitic scars or erosions are to be seen, the diagnosis is manifest.

It is very common in syphilitic epilepsy to find that the attacks are followed by prolonged attacks of paralysis, not due so much to the exhaustion of the centres as to the irritation produced by the gumma or the inflammation which sometimes springs up around it. It is also a noteworthy fact that the paralysis most commonly seen involves the oculomotor, abducens, and patheticus nerves.

The diagnosis of syphilitic epilepsy from the idiopathic form is of the utmost importance, since the ultimate result must be largely governed by the cause. Dowes has analyzed no less than 274 cases in order to discover any useful points in this respect. He insists, as the writer has already done, that epileptic attacks beginning after thirty years of age are almost surely syphilitic, particularly if no history of traumatism or heredity is present. It is also found that, if some degree of mental alienation is present between the paroxysms, it will generally yield to specific remedies. Cyanosis is less frequent, and pallor is more common in syphilitic epilepsy than in the ordinary disease.

The convulsions of an eruptive fever differ from the true epileptic attack very slightly indeed. It is only by the history of the patient and by waiting for developments that we can determine which is which, for as soon as the eruption or high temperature of an exanthem occurs the character of the attack is evident.

Epileptic convulsive disorders may arise owing to the action of a very large number of toxic substances, of which the writer shall here consider only a few, as an enumeration of all of them is manifestly impossible.

Alcoholic epilepsy consists of two distinct varieties produced by overindulgence in intoxicating drinks. In one of these the convulsions are symptomatic of acute poisoning, and come on during a drunken orgy, or immediately after a single large draught of liquor. In the second variety the convulsion does not originate while there is alcohol in the blood, but in the intervals between the attacks of delirium tremens resulting from chronic excessive alcoholic indulgence. Under these circumstances the paroxysms are generally accompanied by hallucinations or by dementia or imbecility. In the alcoholic convulsion the symptoms may closely resemble those of true epilepsy, and not rarely the attack is ushered in by headache, gastric embarrassment, disorders of vision, and excessive tremors, or some similar prodrome which may be looked upon as partaking of the nature of an aura. As a general rule, these alcoholic convulsions occur in paroxysms—two, three, four, or more, one after the other, at intervals of a few minutes. Not only may *grand mal* be closely simulated by alcoholic epilepsy, but simple vertigo or true *petit mal* may exist, either alone or associated with major convulsions. Alcoholic epilepsy is often associated with hallucinations, especially of terror, and not rarely is followed for days by a certain degree of mental disturbance. Rather curiously these cerebral disturbance, result rather in suicidal than homicidal tendencies, which is just the reverse of the insanity following simple epilepsy. It is very important that the reader remember that alcoholism in producing epilepsy very frequently produces a permanent nervous disorder which the withdrawal of the poison will not remove.

The symptoms of an uræmic convulsion will be spoken of further when studying its differential diagnosis in connection with epilepsy.

As some cases of sudden epileptiform convulsions are apt to result in an official investigation as to their cause, and as the character of the treatment of the case before death may influence the question of life and death for the accused very greatly, it is well for the physician to bear in mind that certain drugs when taken in poisonous doses produce well-developed epileptic convulsions. This is particularly true of the so-called cardiac sedatives or depressants, such as aconite, veratrum viride, sabadilla, hydrocyanic acid, and one or two similar substances. Suffice it to say that experimental researches have proved that they act by disordering the cerebral circulation.

The symptoms of epilepsy due to chronic poisoning by lead are chiefly as follows : The man, apparently in his usual health, or

who has had for a few days a feeling of weight in the head, or headache, is suddenly seized with most violent convulsions, which are often fatal, and which during their presence resemble ordinary epilepsy so closely as not to be separated from it. They end in coma, and are separated from each other by intervals of nervousness and disquiet. In some cases one convulsion follows the other so rapidly that death ensues from exhaustion, but in much more rare instances the attacks may resemble Jacksonian epilepsy very closely, and there may be no loss of consciousness. If such a condition occur, it is almost sure to be followed by a more violent fit. The attacks are not preceded by any aura whatever, but previous to the headache, already mentioned, the patient may have had amaurosis, and ophthalmoscopic examination of the eyes may show choked disk and neuritis of the optic nerve. As a general rule, such cases are fatal, but they may recover under careful treatment.

Malarial epilepsy is an uncommon disorder, even in countries and regions which are notoriously malarial, but it has undoubtedly occurred, particularly in the southern part of the United States and in Brazil. The only cases which the writer can find recorded are by American or English observers, namely, Jacobi, Payne, and Hamilton. The latter gives but a passing glance at the subject, and the articles of the others the author has not been able to obtain; so that he knows them solely by reputation. In Hamilton's case, a young man, who had lived for many years in an exceedingly malarious region, had more or less periodic epileptic attacks, attended by great preliminary rise of temperature and intense congestion of the face and head. He was unusually somnolent, and in the intervals frequently suffered from facial neuralgia. Change of the place of habitation and the use of quinine removed the disease entirely.

The differential diagnosis between idiopathic epilepsy, that which is due to demonstrable cause, and the diseases which resemble it, is quite possible in many cases.

Undoubtedly the most similar convulsive condition that we have is that known as hysteria, and the diagnosis of one from the other is as difficult in some cases as it is essential and necessary for treatment and cure. The other conditions with which it might be confused are uræmia, alcoholic epilepsy, tetanus, and syncope. On the following page are arranged all these disorders in a table, which briefly and succinctly shows the different points between them, although, of necessity, it is somewhat arbitrary on account of the lack of

space. Nevertheless, it is hoped that it will be clear enough to be of service, particularly in connection with what the author is about to say.[1]

TABLE OF DIFFERENTIAL DIAGNOSIS OF EPILEPSY FROM HYSTERIA, ETC.

Signs.	Epilepsy.	Hysteria.	Uræmia.	Petit mal.	Alcoholic epilepsy.	Tetanus.	Syncope.
Apparent cause,	None.	Emotion.	None.	None.	None.	None.	Mental shock.
Aura or prodromata,	Generally present, but short.	Globus hystericus; palpitation; choking.	Headache, vomiting, and dyspepsia.	Faintness and dimness of vision.	Tremors.	Nervousness.	Not so well defined as in epilepsy.
Onset,	Sudden.	Often gradual.	Often gradual.	Sudden.	Sudden or gradual.	Gradual; begins in jaw.	Sudden or gradual.
Scream,	At onset and sudden.	During attack.	Frequently none.	Frequently none.	May or may not be present.	None.	None.
Convulsion,	First tonic, then clonic.	Rigidity more pronounced, with more aching.	Rigidity generally absent.	No rigidity.	Movements more clonic than tonic.	Always tonic.	None.
Biting,	Tongue.	People, tongue, lips, and hands.	Tongue.	None.	Rarely.	None.	None.
Micturition,	Frequent.	Never.	Never.	Rarely, except when bladder is affected.	Rarely.	Sometimes.	Never.
Defecation,	Occasionally.	Never.	Never.	Never.	Rarely.	Rarely.	Never.
Talking,	Never.	Frequent.	Muttering.	Never.	Never.	Never.	None.
Duration,	A few minutes.	Generally many minutes.	From a minute to hours.	Momentary.	May be prolonged.	Hours.	Indefinite time.
Consciousness,	Lost.	Generally preserved.	Lost.	Not lost always, but clouded.	Lost.	Preserved.	Lost.
Termination,	Spontaneous.	May be induced by shock.	Spontaneous.	Spontaneous.	Spontaneous.	Spontaneous.	Gradual, with no somnolence.

The very irregularity of true epilepsy makes it extremely difficult to give clear and well-defined outlines of it against another disease, particularly when we remember that epilepsy and hysteria often go hand in hand.

By far the most important differential point between the two disorders just named, when not complicated with still another disease, is the character of the movements. As already pointed out, in epilepsy they are typically at variance with those of daily life, while in hysteria they are almost equally typical of ordinary muscular contractions, or, in other words, are more purposive in character; and frequently there is prolonged tonic contraction of the muscles, giving

[1] This table is taken from the author's essay on Epilepsy, the prize essay of the Royal Academy of Medicine in Belgium, January, 1889.

rise to the assumption of positions which bear more or less resemblance to normal attitudes. In hysteria, also, consciousness is impaired sometimes, but never so completely as in true epilepsy. Indeed, most commonly the individual knows all that goes on around her, for, while she may give no sign of consciousness by words or looks during the attack, she may afterward be able to narrate all that has occurred. Less commonly, however, a condition known as automatic consciousness exists, in which, during the paroxysm, the patient understands all that is said, but forgets everything on the return to quietness.

The fact that the patient is a female cannot be regarded as affirmative evidence of hysteria in the least, but the condition occurring in a male may be taken as fairly positive evidence of its being epilepsy; and yet it should always be remembered that males may suffer from hysterical attacks.

The movements of the hysterical patient after the tonic condition has passed away are as clonic as those of the epileptic, but still possess some purposive characteristics, and are not so bizarre as are those of the true disease. Thus the head, arms, and legs are struck with evident endeavor against the floor or surrounding furniture. Another point, which, when it occurs, is very distinctive, is the onset, toward the close of a hysterical convulsion, of a second stage of tonic spasm, such as occurred at the beginning. It will be remembered that this does not occur in epilepsy, although it must be borne in mind that in cases of the "status epilepticus" the rapid onset of another attack may show a second tonic stage. This can be separated, however, by the fact that it is followed by clonic movements, whereas the secondary tonic stage of hysteria is usually followed by relaxation and temporary recovery.

In the secondary hysterical tonic contractions emprosthotonos and opisthotonos may occur, and are even more rigid in their character than they are in the first attack, in some cases. Finally, too, in hysteria, some peculiar emotional position is often assumed, as of the crucifix or of intense grief, or, perhaps, immoderate laughter is indulged in, with corresponding movements of the trunk. If the patient is quiet at this time a smile may float across the face, while the eyes, with a look of pleasure, pain, or entreaty, may seem to be gazing at some object very far off. In some very well-developed cases the expression of pleasure is followed by a look of pain, with painful movements, or an appearance of intense voluptuous entreaty,

with sensual venereal desire evidenced by gestures. Great terror may be present, and, as the scene constantly changes, the woman is now joyous, now mournful, now scolding, now praising her attendants or herself. Such is the history of a fully developed attack of hysteria.

Hysterical convulsions in their fully developed form are rarely seen among Americans, Germans, Belgians, or corresponding races, but are very frequently observed by French practitioners of medicine.

In France there can be no doubt that the tongue is commonly bitten in hysterical convulsions, and that frothing of the mouth is frequently present; but in other countries this symptom may be regarded as indicative of epilepsy rather than hysteria. Doubtless the inexperienced reader will say, upon comparing these symptoms with those which were given as occurring in epilepsy proper, that the two disorders are easily separated from one another; but the author would insist most strenuously upon the fact that in both cases he has given only the most typical characteristics of the diseases, and he repeats that all cases are not by any means so well defined. He would also remind the reader that the chief difficulty in making a diagnosis lies in the fact that frequently it must be made without any previous history of the case, as when a patient is brought into a hospital, in a fit, for treatment. Where the history is obtainable or where the diagnosis can be put off until the case may be studied, the question becomes more easily solved.

If a large number of patients suffering from these hysterical attacks be questioned between times, it will be found that the so-called globus hystericus becomes an almost constant precursory symptom of an attack; and if the relatives be questioned, it will often appear that they have noticed that the fall to the floor is more gentle than in true epilepsy; but this is not always so by any means. Again, the expression of the face in hysteria is, between the attacks, often very characteristic, and the surrounding atmosphere of the patient seems, even to the inexperienced, to breathe hysteria. Very commonly areas of anæsthesia and hyperæsthesia occur in these patients, and are of all degrees of intensity and limitation. Search for them generally shows their presence after attacks of convulsions, but they may exist from one attack to the other, or develop spontaneously. In nearly all cases these areas are unilateral, and may extend entirely over one-half of the body, the line of demarcation of the anæsthesia or hyperæsthesia from the sound area

being clearly and abruptly defined, generally at the median line of the front and back of the trunk. (See chapter on the Skin; that part dealing with anæsthesia.) It will be called to mind that such conditions are very rare in true epilepsy. Hallucinations are far more common after the fit in hysteria than in epilepsy, and sometimes they even occur during the attacks. They are always associated with the mental state; if terror is present, rats or disgusting objects are seen, and, according to Charcot, are generally seen on the side which, during the intermissions, is anæsthetic. The pupil is more mobile in hysteria than in epilepsy, but may be contracted, normal, or widely dilated.

The following table gives, in as brief a manner as possible, the differential diagnosis between epilepsy and hystero-epilepsy, and is founded on a lecture by Professor Charcot, delivered at the Salpêtrière.

True Epilepsy.	*Hystero-epilepsy.*
Aura short.	Aura extremely prolonged.
Cry is violent.	Cry is more moderate and prolonged.
Spasms first tonic, then clonic, then followed by stertor.	Ataxic contractions, extension of limbs, turning of head, clonic movements, slight stertor.
Sometimes after fit delirium or violent impulse or mania.	Bizarre contractions, no delirium, may be hallucinations.
Mental power is lost.	Mental power preserved.
No emotional attitudes.	Emotional attitudes.

A very useful differential point, strongly insisted upon by Charcot and Bourneville, is that in true epilepsy there is generally a very considerable rise of temperature during an attack, while in hystero-epilepsy the temperature remains normal or only slightly raised.

In the diagnosis of true epilepsy from convulsions of a hysteroid character it is well for the physician to remember that the proportion of the two conditions in frequency of occurrence is, according to Gowers, 815 to 185 in every 1000 cases.

The differentiation of epilepsy from *uræmia* is much more readily carried out, for there is usually a previous history of symptoms pointing to renal trouble, as, for example, some œdema, or somnolence, or mental apathy, for some days or hours before the attack. Of course, in such cases recourse may be had to the ordinary tests for such conditions of the urinary organs as are generally found where uræmia exists; but it is to be remembered that epilepsy and kidney disease may exist hand in hand, and that for this reason the prognosis and diagnosis are to be carefully formed and given. If in a given case a prolonged history of dyspepsia, of frequent vomiting,

occasional attacks of asthma, and failure of general health is found to be present, the correct diagnosis probably will be uræmia. The preservation or loss of consciousness in uræmic convulsions is variable. Generally, if the convulsion is widespread and severe, the intellection is lost; but if it be only a slight attack, consciousness may be preserved. So long ago as 1840 Dr. Bright described cases of uræmia, on the other hand, in which furious convulsions occurred without loss of consciousness, and Roberts has reported similar instances.

Just here the author may remind the reader that not more than thirty years ago some physicians of very high standing believed epilepsy to be due entirely to uræmia. Thus Sieveking firmly believed in this theory, and reported a case in support of his views. Fatal uræmia may also occur in a patient whose urine is apparently normal; and, in a large number of cases of chronic contracted kidney, albumin may be absent from the urine for long periods of time. The specific gravity of the urine should be carefully noted, and in very doubtful cases careful estimations of the urea be made. If the specific gravity is constantly below 1.010, the kidney will nearly always be found contracted unless diabetes insipidus exists. Tests of the urine passed at different times of the day should always be made. Another means of testing the integrity of the kidney is to administer iodide of potassium and study its elimination. It is affirmed that, after a full dose, this drug can in an hour be readily recognized in the urine by adding nitric acid and then starch; but when contracted kidney exists the iodide fails to appear or is excreted only in very small quantities. The temperature of the body may also be used to differentiate between uræmia and epilepsy. In 1865 Kien called attention to the fact that even when uræmic convulsions are most violent they are accompanied by a fall of temperature of as marked a character as the rise noted in respect to epilepsy. Since then this has been confirmed by Roberts, Hirtz, Hutchinson, Charcot, Bourneville, and Teinurier.

The diagnosis between *puerperal eclampsia* and epilepsy consists chiefly in the acuteness of the attack, and the fact that with no previous convulsive history a woman becomes suddenly convulsed during the puerperal state. This is not a place for the discussion of the identity of uræmia and puerperal eclampsia, although we believe that uræmia is generally responsible for the nervous disturbance. If the convulsions are uræmic, the temperature, according to the investigators just quoted, should fall; and according to Bourneville, puer-

peral convulsions are distinctly separated from those of uræmia by reason of the fact that the temperature rises with great rapidity in the very beginning of the convulsions and there remains with great steadiness. The condition of bodily temperature can, therefore, be used to differentiate puerperal eclampsia and uræmia.

It is unnecessary to state once more that *petit mal* is but a variety or modification of *haut mal.* Nevertheless, it is useful to be able to separate it somewhat from the more severe form of the disease in the attempt to form a prognosis.

Some suppose that *petit mal* may be designated as consisting of one or two of the chief symptoms of epilepsy proper, and others have thought that the preservation of consciousness was the chief dividing-line between it and fully developed epilepsy. The last idea is certainly incorrect, but it is impossible to give any outline which will absolutely separate the two conditions, so far as symptoms go. An important and useful point first discovered by the celebrated neurologist, Weir Mitchell, is that, whereas the inhalation of amyl nitrite stops true epilepsy, the use of this drug increases the severity of an attack of *petit mal.*

Alcoholic epilepsy occurring during an attack of *mania a potu* is, of course, easily diagnosed, and the general appearance of the patient, combined with his history, suffices to make the physician's decision. The movements are more clonic than tonic, and often are lacking in force. There is, however, no constant distinction between the symptoms applicable to all cases. Generally one seizure of alcoholic epilepsy follows the other every few minutes until three or four have taken place, when the paroxysms cease. It is not to be forgotten that alcohol may produce all degrees of epilepsy, from the mildest *petit mal* to the most severe paroxysms, and it is also to be remembered that hallucinations of terror are very commonly present.

There may be an aura in alcoholic epilepsy quite as marked as in the true disease.

The separation of *syncope* from epilepsy is one of the easiest tasks imposed upon us. The color of the face, the weakened heart-beat, sudden loss of consciousness, and the general appearance aid us here very much.

The separation of epilepsy from hemicrania has been very well written of by Silva. He thinks that epilepsy begins in childhood before puberty, most commonly, while hemicrania comes on after puberty; and that the attacks of hemicrania decrease in violence and

frequency as age increases, while the contrary rule applies to epilepsy. These views are in accord with those of Strümpell and Wagner.

The diagnosis of *lead epilepsy* from the idiopathic varieties is somewhat difficult, if the patient is seen for the first time during an attack; but the ordinary methods of determining chronic lead-poisoning are, of course, of equal value here. The blue line on the gums may be present, and, if so, the diagnosis is almost certainly lead-poisoning; but its absence is no proof that lead is not present. The administration of iodide of potassium also will so increase the elimination of the poison as to benefit the case and render it more easy to recover lead from the urine.

The history of exposure to lead in any form is, of course, exceedingly valuable evidence, but it should not be forgotten that in many cases this history is wanting. Thus, the poison may be derived from a hair-dye, or cosmetic, or from water which contains lead from pipes, or from an endless line of similar hidden and obscure sources. Amaurosis may be present in some cases, or optic neuritis with atrophy may occur. Where double wrist-drop is present the diagnosis is much more easy.

It is exceedingly important to differentiate between those convulsions which arise from the uræmia brought on secondarily by an action of lead on the kidneys and those which are due to a direct action on the brain. This may be difficult from the mere symptoms presented, but there are some points of difference. In the first place, the convulsion of uræmia is, as a general rule, not so violent in its movements nor so sudden in its onset. It is generally preceded by a few days of somnolence, or weeks of gastric disorder and headache, while lead epilepsy is generally sudden or preceded by cephalalgia by only a few days or hours. Again, examination of the urine in uræmic convulsions will show a decreased amount of urates in proportion to the quantity of urine passed, while in plumbic epilepsy just the reverse will be true, unless the kidneys are affected *pari passu* with the cerebrum. If albumin be present, uræmia is pointed to; but if the urine has a low specific gravity and is passed in large amounts, the indications are that there is chronic contracted kidney, which may or may not be the cause of the nervous disturbance.

Before closing this portion of this chapter the writer must bring forward the points to be used in differentiating epilepsy from those attacks simulated by malingerers. Often this is most difficult; and

it is related of Fournier that, after his expressing an opinion that a man could always tell them apart, one of his assistants threw himself to the floor on his next visit in a pretended attack, whereupon Fournier, completely misled, exclaimed, "Poor M—; he is epileptic!" upon which the assistant, smiling, arose to his feet and confuted the statement.

Very serious injuries are sometimes submitted to by these persons to carry out their designs. The points to be looked into are: The condition of the pupils, which, in the simulated attack, always react normally; nor can the corneal reflexes be held back; the color of the face is rarely changed; and the thumbs are rarely flexed as they should be. Marc has pointed out that in malingerers the bystander can readily straighten the thumbs out, and that they remain so; whereas in epilepsy they instantly become flexed again.

Suggestions, as to movements, are sometimes followed by malingerers, and the movements generally lack the bizarre character so typical of epilepsy.

If tobacco or ammonia be held to the nose of the fraud, he generally is forced to disclose his true nature.

The fact that in malingerers there is no rise of temperature may also serve as a differential point.

Convulsions appearing in infants or young children may result from injuries to the brain in birth, to the presence of growths, or to other distinct cerebral causes and irritation of the alimentary canal. In these cases they may be reflexly produced. Certainly they often arise from the reflex irritation produced by teething in children entirely free from rickets and from gastro-intestinal irritation due to the ingestion of improper foods. Whether adherent prepuce and other causes of peripheral irritation ever result in convulsive seizures is a matter of doubt, some authorities believing that such causes are frequently present, while others deny their existence. The author believes that given a child with a distinctly neurotic temperament and a marked source of peripheral irritation, convulsions are produced. Stevens asserts that insufficiency of the ocular muscles very frequently causes epilepsy, and he is not alone in this belief. Certainly in cases in which such possible causes of nervous excitation exist the physician should remove them as his first attempt at treatment.

There is one variety of infantile convulsive seizure due to meningitis which is in itself often tubercular and associated with retraction of the head and squint; and another variety in which the

symptoms very closely resemble those due to actual meningeal lesions, but in reality are quite independent of them. This condition has been called "pseudo-meningitis" or "hydrocephaloid disease," and is seen in young infants generally after attacks of severe diarrhœa. The fontanelle is depressed, the child is somnolent or comatose, and fever may or may not be present. The prognosis in the first class of cases is very bad. In the second class it is bad enough, but recovery quite often occurs if the treatment generally employed in the first class is set aside and a highly nutritious and supporting treatment is instituted.

If a child suddenly develops symptoms of acute meningitis and has delirium, rigidity of the neck, and the major manifestations of the disease, the lungs should be carefully examined for croupous pneumonia, as this disease in children very often causes these cerebral or meningeal symptoms. Even in the adult maniacal delirium and rigidity of the neck may be present in croupous pneumonia.

Convulsions, which are epileptiform, sometimes occur in the latter stages of Addison's disease.

Tetanic Convulsions. The convulsions which are of spinal origin, namely, those that are tetanic, are the result of *tetanus* or the *ingestion of strychnine* in poisonous dose, or its fellow ignatia, and sometimes are due to hysteria. The diagnosis is aided by what has been said in respect to the symptoms of hysterical convulsions in the last few pages, and finally by the discovery of the hysterical stigmata, or the signs manifested by the skin, and when examination can be made between attacks, of the eyes (see chapters on Skin and Eyes).

Tetanus convulsions and strychnine-poisoning are to be separated from one another by the fact that in tetanus the locking of the jaws comes first, while in strychnine-poisoning it comes last. The convulsions of tetanus rarely, if ever, completely relax, while those of strychnine do have periods of relaxation. There is a different history in each case. In one, perhaps, of an injury, as of a nail run into the foot; in the other, of a dose of poison having been swallowed.

The differential diagnosis between strychnine-poisoning and hysterical convulsions is more difficult. The convulsions are rarely so persistently tonic in hysteria as in strychnine-poisoning, and the peculiar expression of the hysterical face is often seen in this disease. The history of the patient, if obtainable, will throw much

light on the case and aid very materially in the separation of the two conditions.

When a patient is seized with sudden and symmetrical tonic spasms of the hands, extending to the upper arms and shoulders, so that the fingers are flexed at the metacarpo-phalangeal joints and extended in the phalangeal joints, while the lower arm is flexed and the legs extended, while the toes are flexed, the condition is one of *tetany.* It is most commonly seen in hysterical cases and has no relation to true tetanus. Pressure on a nerve-trunk or bloodvessel will often produce an attack in such persons. This is sometimes called "Trousseau's symptom." The pressure must be applied for several minutes in some cases, and the best place to apply it is the bicipital sulcus or the crural sulcus. It is not a constant symptom, but pathognomonic if found. Another equally useful diagnostic sign is called Chevostek's symptom. This consists in the fact that the muscles are irritable, so that when they are tapped by the finger-tip, or hammer, contraction results. The prognosis is favorable, but recurrences are frequent. It occurs most commonly in males up to twenty years, and may occur as early in life as two to four years of age.

Convulsions limited to a few muscles or more widespread in character may appear as symptoms in *acute yellow atrophy of the liver;* but the peculiar symptoms of this disease render easy the diagnosis of their cause.

Typical epileptiform convulsions are the most constant symptoms of *hydatid cyst* in the cerebral cortex, but the diagnosis of this condition is impossible unless from a history of probable infection.

Convulsions may also arise from *hæmatoma* of the dura mater (internal hemorrhagic pachymeningitis), but the diagnosis from those due to cerebral hemorrhage is practically impossible.

General violent convulsions have also been seen quite frequently in nervous patients during the paroxysmal pain of *gall-stone colic,* and they also sometimes usher in the acute poliomyelitis of childhood and the infectious diseases.

Epileptiform convulsions may come on in adults as the result of *multiple sclerosis,* and they are very commonly seen in sunstroke when the patient is first attacked.

Curiously enough, severe convulsions have been known to follow *irrigation of the pleural cavity* after aspiration, and they may also be seen in young children suffering from whooping-cough at the time of the paroxysm.

Spasms.

General spasms, in distinction from convulsions, are represented by chorea in its various forms, and by saltatoric and palmic spasm, paramyoclonus multiplex, and the occupation-neuroses. There are other localized spasms from nervous disease, such as facial spasm and wry-neck, athetosis, and post-hemiplegic chorea. Some of these conditions will be found discussed in the chapter on the Hand and Arm and that on the Face and Head.

When a patient is afflicted more or less constantly and more or less universally by disordered, irregular, jerking movements which throw the part or parts affected into unusual positions, which are not, however, maintained even for a moment, the condition is probably *chorea minor*. Often the speech is seriously disturbed by reason of the choreic movements of the lips and tongue or jaws, and some loss of power may be manifest in certain muscles. This true chorea or St. Vitus's dance may affect the whole body or only one arm or leg, but generally it is diffused. Commonly it ceases at night when the child sleeps, but it often persists day and night, and then becomes a serious malady, because of the exhaustion produced. It often follows fright, prolonged bad weather, and other causes which may upset the nervous balance of the child. Chorea is so characteristic in its manifestations that it can be readily recognized in most cases; but it sometimes has to be separated from disseminated sclerosis, progressive muscular atrophy, hysteria, and Friedreich's ataxia. The movements in disseminated sclerosis are, however, fine muscular tremors, instead of minor jerking movements; and there are present nystagmus and scanning speech in sclerosis, but not in chorea. Again, in progressive muscular atrophy there is fibrillary muscular tremor, but not twitching of a marked form, and the muscles are wasted. In hysteria the muscular movements are rarely choreic, and the presence of changes in the color-fields and the other stigmata of hysteria (see chapter on Skin and Eyes) renders a diagnosis of the latter condition easy.

Friedreich's ataxia is to be separated from chorea by the scanning speech, scoliosis, slow inco-ordinate movements, and the family history of the disease.

Rarely, when there is some paresis with chorea, the patient may present symptoms of acute poliomyelitis; but the paralysis in the

latter affection is more marked, and there are no movements in the affected muscles, such as occur in chorea.

Chorea insaniens is a violent form of ordinary chorea associated with mania, which is not to be confused with choreic movements occurring in the choreic insane.

Choreic movements sometimes come on in the aged, and must be separated from paralysis agitans and senile trembling. This is possible by the fact that in paralysis agitans the movements are tremors, and there is loss of power with the peculiar facial expression ("Parkinsonian visage"). Senile trembling is usually an affection limited to the head, and consists in a tremor and not in marked twitching. (See chapter on Hand and Arm; part on Tremor.)

A rare form of chorea has been called *Huntington's chorea.* It occurs in adults about the age of thirty to forty years. It is hereditary; that is, there is generally a history of the same trouble in the ancestors of the patient, and finally as it progresses psychical disturbances ensue. Irregular movements first appear in the hands, which movements become markedly inco-ordinated, the arms are thrown about in excessive and rapid jerkings, and when the infection involves the legs a characteristic gait is developed of a dancing or "hop, skip, and jump" character. Sometimes, early in the malady, the movements can be controlled by the will. The face passes through slowly formed grimaces, and the gait may be staggering. The speech becomes indistinct, and enunciation is not clear. Finally, dementia closes the scene. The movements of Huntington's chorea are not sudden as in true chorea; it is a disease of adult life, and mental disturbance is a prominent symptom. These facts separate it from ordinary chorea.

When the patient involuntarily bends over in a profound bow the cause of his movements may be rhythmical contraction of his abdominal muscles, producing the so-called *salaam convulsions* or chorea major.

A still more rare malady is *electric chorea* or "Dubini's disease," in which the muscles of the arm and then the leg on the same side are affected with a sudden muscular spasm or shock, such as is produced by the electrical current. Wasting of the affected muscles, loss of faradic irritability, occasional epileptic convulsions, and rarely elevation of temperature come on. The disease is a fatal one, and generally occurs in Italy in malarial regions. Under the same name

of electric chorea Bergeron has described a state of rhythmical muscular spasm which usually ends in recovery.

When a condition of clonic muscular spasm affecting the trunk, limbs, and perhaps the neck, is present, the hands and toes being uninvolved, as a rule, the possibility of the presence of *paramyoclonus multiplex* is to be considered. The spasms in this rare disease are bilateral and occur at intervals, often only on an attempted movement or speech. So violent are the muscular contractions in some cases that the patient may be thrown to the ground or, if in bed, to the floor. These movements may vary from three or four to 120 per minute, but are generally about fifty per minute. The symmetrical bilateral involvement, the fact that the movements are not choreic in character, and that the patient is a male, are to be remembered in making the diagnosis. The ultimate prognosis is favorable unless the movements are so constant as to cause exhaustion. Care must be taken not to confuse hysterical movements with this condition. The bilateral movements which affect only the larger muscles, and the fact that paramyoclonus multiplex is nearly always seen in the male, separate it in part from hysteria, while the hysterical stigmata when they are present will point to hysteria as the cause of the disorder.

Sometimes a patient will be met with in whom, when he attempts to stand, the leg muscles first become rigid and then are thrown into violent contractions, which cause him to jump up and down, or he may be thrown to the floor. This condition is called *saltatoric spasm* or "jumpers." It is to be separated from the condition of the legs seen in lateral sclerosis of the cord by the fact that in the latter disease the legs become spastically stiff on attempting to use them, from Huntington's chorea in that voluntary movements with the hands may be performed perfectly, and from chorea minor by the absence of small inco-ordinated twitchings.

Such a patient will often act on suggestions or in imitation of the acts of other persons or of animals.

Some writers confine the term "saltatoric spasm" to those cases which possess no imitative features. In such cases the disease is far more moderate in its manifestations.

Quite distinct from these clonic spasms of the muscles brought on by attempted movement is that in which the muscles become tonic on attempted movements. At first they are stiff and slow in their movements, but ultimately develop a tonic spasm, so that

walking is at first almost impossible, but the limbs limber up on exercise. This is a rare affection, called *Thomsen's disease*, or one of the forms of *myotonia congenita*. (See chapter on Feet and Legs.)

Forced gyratory movements of the body are sometimes seen as the result of a lesion of the middle peduncle of the cerebellum.

CHAPTER V.

VOMITING, REGURGITATION, AND THE CHARACTER OF THE VOMIT.

Due to uræmia—Cerebral lesions—Intestinal obstruction—Peritonitis—Cholera—Gastric disease—Hepatic disease—Poisons—The appearance of vomit.

VOMITING is the act by which the contents of the stomach are forcibly expelled from this viscus through the cardiac orifice, the œsophagus, the pharynx, and mouth. The vomiting-centre in the medulla oblongata gives rise to the necessary nervous impulses and is provoked to this by direct stimulation or by reflex irritation. Thus, in uræmia the vomiting sometimes encountered is the result of irritation of the centre by some unknown poison, and when apomorphine is given the centre is also stimulated. Centric vomiting is also caused by the administration of anæsthetics, notably ether and chloroform. On the other hand, gastric, intestinal, or other abdominal disorder may reflexly produce very persistent emesis, and for these reasons vomiting is of considerable diagnostic importance.

Vomiting being produced by many maladies is a symptom frequently met with. It occurs with a certain degree of constancy as a complication or symptom of uræmia, diabetes, apoplexy, brain-tumor, brain-abscess, Ménière's disease, tubercular meningitis, hysteria, intestinal obstruction from all its various causes, gastric and intestinal indigestion, gastritis, gastric ulcer, gastric cancer, peritonitis, nephritic colic, hepatic jaundice, hepatic colic, in cholera, yellow fever, and a host of other ailments.

The vomiting of *uræmia* may be one of the earliest manifestations of renal disease, and its presence, when persistent in the absence of local gastric or other causes, should always lead to an examination of the urine, since valuable time may be lost if the patient is considered to be suffering from some slight indiscretion in diet. Its association either as a preceding, concomitant, or consequent symptom of convulsions renders a diagnosis of uræmia probable, while a history of uræmic amaurosis, colliquative diarrhœa, and failure of the general health will be very important points in reaching a decision. No pathognomonic symptom of uræmic vomiting exists unless we consider the urinary evidence a symptom, but in some cases the vomited

matters smell strongly of carbonate of ammonium, resulting from the decomposition of the urea, which has been eliminated from the blood into the stomach by the gastric mucous membrane. Uræmic vomiting is, therefore, not only due to centric irritation by a poison in the blood, but to irritation of the stomach by the urea which is excreted into it. Diabetes comparatively rarely produces vomiting by the toxæmia which it causes, but in any case the urinary examination and polyuria decide the diagnosis.

When vomiting results from *cerebral hemorrhage, embolism,* or *thrombosis,* the focal or hemiplegic symptoms characteristic of apoplexy are present. Possibly the vomiting is more indicative of hemorrhage than of plugging of the vessel. A sudden attack of vomiting in a previously healthy man of advanced years or in one who is young, but has a specific history, should raise the question as to the possible presence of one of these lesions; provided, of course, that ordinary gastric disorder is not present as a cause. The vomiting due to *cerebral tumor* is generally preceded by the characteristic severe and constant headache, vertigo, a slow pulse, impaired memory, and sometimes by epileptiform convulsions. Further than this, the important diagnostic ocular symptom "called choked disk" of the optic nerve is to be sought for, and if found is of great positive value. Tumor of the brain, if near the base, often causes, too, involvement of the various cranial nerves. The vomiting of cerebral tumor is independent of taking food, and commonly comes on early in the morning, thereby differing from some of the forms of vomiting due to gastric disorder. The vomiting arising from *cerebral abscess* has symptoms precisely like those just named, so that a differential diagnosis is almost impossible. The history of injury or of an infectious process producing a secondary brain-abscess may point to this cause of the vomiting; the real points of difference are that in abscess choked disk is rarely seen, fever is commonly present, and the cranial nerves generally escape. When *purulent meningitis* produces vomiting it may be impossible to tell whether this symptom is due to it or to an abscess, as the purulent collection may be localized. Vomiting sometimes results from *profound cerebral anæmia* of an acute type due to hemorrhage, in fainting or in chronic anæmia, as in chlorosis. Generally, however, the symptom is only a constant nausea. The presence of great pallor and other evidences of anæmia aids in the diagnosis, but it must not be forgotten that some severe anæmias are accompanied by febrile movement and by marked

choked disk, which should not mislead the physician into a diagnosis of cerebral tumor.

When vomiting is due to *cerebellar tumor* the diagnosis is aided by the presence of vertigo, the peculiar staggering gait, and finally by evidences of choked disk, on ophthalmoscopic examination, with disordered vision.

The vomiting of *meningitis* is quite frequently an early symptom, but it also often occurs later in the disease, and is caused by the meningeal irritation, and not by any condition of the stomach, unless that viscus has been disordered by the unwise use of drugs. This form of vomiting can nearly always be separated from that due to other causes by the excessively severe headache, chiefly of an occipital type; by the pain in the nape of the neck and in the spine; by the rigidity of the dorsal muscles, so that opisthotonos may be caused in severe cases; and, finally, by the disordered functions of the cranial nerves, as a result of which there are found trouble in the oculomotor nerve, strabismus, double or single ptosis, slowly reacting pupils, which may be unequal, nystagmus, and sometimes facial contractions due to involvement of the facial nerve.

Vomiting due to *acute miliary tuberculosis* often comes on at the very onset of the malady, and is associated with obstinate constipation, or, on the other hand, active diarrhœa; but the fever, the very rapid pulse, the wasting of the patient, the possibly present physical signs of tuberculosis of the lungs, and, very important, the peculiarly severe dyspnœa, for which no adequate cause can be discovered on physical examination, all point to the general infection. If a skilful examination of the eye can be made with the ophthalmoscope, the choroid may be found to be studded with tubercles.

The reflex forms of vomiting are very numerous, and depend chiefly upon organic and functional disorders of the abdominal viscera. In some of these conditions vomiting is of little importance, except for its gravity if the patient is exhausted; in other words, it is simply a disagreeable symptom. In others the symptom vomiting is of considerable diagnostic value as indicating the grave mischief which produces it. One of the most important of the latter conditions is *intestinal obstruction*, whether it arises from intussusception, constrictions by bands, volvulus, or impactions. In *intussusception* vomiting is practically a constant symptom, occurring with the sudden pain, or, at times, even preceding it. In children it continues till shortly before death, and is rarely feculent.

In the adult, and in the chronic form, there may be complete absence of vomiting, though this is certainly exceedingly rare. Leichtenstern takes exception to the statement that the seat of obstruction is indicated by the period at which vomiting is developed.

The ileum-invagination is most frequently accompanied by early vomiting, not because of its seat, which is usually but little removed from the ileo-cæcal valve, but because it is commonly obstructive. The vomiting, both in time of development and in nature, will depend, not upon the seat of the trouble, but upon the presence or completeness of obstruction, and may be early if the obstruction is absolute in the sigmoid flexure, and feculent if the bowel is occluded in the upper part of the ileum.

The pain is usually sudden, violent, diffuse, or, if localized, usually placed in the ileo-cæcal or umbilical region. After a few hours in children, a much longer interval in the adult, the pain ceases, often as suddenly as it commenced, and there is an interval in which there is little to suggest that the pathological condition still continues. This is followed by a return of the pain, the paroxysms becoming more violent and prolonged, the intervals less marked as the disease progresses, or in the adult, if it passes into the chronic form, and intervals even of days may elapse between the paroxysms. The pain is frequently accompanied by tenderness, but this is an exceedingly variable symptom, and at times pressure seems to relieve the pain.

Blood-stained mucous evacuations are a symptom of intestinal obstruction which, in children, is rarely wanting. Of 108 cases of invagination in the first year of life this symptom was absent in but four. It occurs within a few hours of the first attack. At the first the discharge is of blood-stained feces ; later, if obstruction is developed, of blood and mucus, and is usually exceedingly offensive. In children diarrhœa is common throughout the whole course of the case. At times, following complete constipation and feculent vomiting, there will suddenly appear copious evacuations from the bowel, mingled with blood, and in which may be found evidences of the necrosed intussusception. Where this slough is extensive it may be lodged in a lower portion of the bowel and cause fatal obstruction.[1]

In connection with the muco-sanguinolent evacuations the tenesmus or straining is a symptom so common that it is of some diag-

[1] For much information on the subject of intestinal obstruction see the Fiske Fund Prize Essay of the Rhode Island Medical Society for 1890, by Martin and Hare.

nostic import. That it is not dependent upon the character of the evacuation is shown by the fact that it is present in cases of complete obstruction. Brinton has shown that this symptom is seldom developed except in the ileo-cæcal and colon invaginations.

A much rarer condition, and one which Leichtenstern ascribes to the secondary effect of intense tenesmus, is a patulous condition of the anus due to paralysis and dependent upon invagination of the descending colon and rectum. This is never produced by invagination of the ileum.

Leichtenstern's statistics show that a tumor can be felt either through the parietes or by rectal examination in 52 per cent. of all cases. In the first year of life this most important diagnostic sign was present in 63 per cent. The tumor is usually felt in the left iliac region, or by the finger passed into the anus. The ileo-cæcal invagination is most frequently accompanied by demonstrable tumor; the ileum-invagination exhibits this symptom with less frequency.

Many authors have noted that the tumor varies in size and consistency from time to time, now, during an acute paroxysm of pain, being hard, knotty, and plainly perceptible, shortly afterward eluding the most careful search. Duchaussoy has described two distinct movements which can often be perceived in the tumor, namely, the erectile and the vermicular motion.

Distention of the abdomen is not of great significance, because it is often absent. In children especially it may not appear at all, or only just before death. In adults, in whom obstruction is more common, it may become as well marked as in obstruction from any other cause.

Dance calls attention to an inequality in the shape of the abdomen dependent upon the meteorism, and in view of which he states that the seat of obstruction can often be inferred. But few authors, however, have been able to profit by his observation.

In the chronic form of invagination the symptoms are less violent in onset; there may be nothing more characteristic of the attack than recurring paroxysms of pain, meteorism, and obstruction, with symptoms of intestinal stricture constantly manifesting themselves. These cases terminate either in cure by reduction or by extrusion of a slough, or perish from exhaustion, inanition, or in the course of an acute attack. In over one-half of the recorded cases a correct diagnosis was not made.

The additional symptoms upon which a diagnosis of vomiting from intussusception is to be based are the acute onset of colicky

pain, and its intermittent character; passages from the bowels containing blood and mucus; the presence of a tumor, commonly in the left iliac region, or felt through the anus, varying in size and consistency from time to time, with an erectile or vermiform motion; and the ordinary obstruction-symptoms. The diagnosis is further confirmed if there are violent peristalsis and tenesmus, and if these symptoms occur in an infant.

According to Leichtenstern, Bryant, and others, 40 per cent. of all cases of intestinal obstruction are due to intussusception, and this condition is most common in the first year of life, after which it becomes more and more rare until the fortieth or fiftieth year, when it increases in frequency. The prognosis is bad, the mortality varying from 73 to 90 per cent. unless early surgical relief is given.

Internal strangulation by bands occurs in from 25 to 30 per cent. of the cases of obstruction of the intestine, and affects males most commonly between twenty and forty years of age. In 133 out of 151 cases the small intestine was involved. The typical symptoms are as follows:

1. Sudden, agonizing pain, constant, and located about the umbilicus, with paroxysmal increments.

2. A rapid, weak pulse and subnormal temperature. This is nearly constant in the early stages of the attack; later on, when local or general peritonitis develops, the temperature and pulse may assume the features characteristic of inflammation.

3. Vomiting. First of the contents of the stomach, then of bile, and, finally, in a large percentage of cases, of feculent matter. The feculent vomiting rarely appears before the third day, and in cases running a very acute course death may ensue before this symptom has time to develop. The vomiting is constant and gives no relief to the patient.

4. Constipation. Exceptionally there may be one or two passages representing the contents of the bowel below the seat of obstruction; after that the constipation is absolute, not even flatus passing by the anus. Treves has suggested that the evacuations sometimes observed toward the termination of the case, and not due to the relief of obstruction, may be dependent upon the beginning of peritonitis.

5. Tympanitic distention. Where there is a large segment of gut involved in the strangulation its early distention may give rise to a localized abdominal enlargement, which is exceedingly suggestive as

to the cause of the attack. In general, the meteorism is not marked except when peritonitis supervenes.

Since in the large majority of cases the obstruction is localized in the lower portion of the small intestine, the primary distention will be observed in the middle abdominal region—*i. e.*, the epigastric, umbilical, and hypogastric areas. Laugier claims by this symptom to locate the obstruction with some certainty.

The violent peristalsis and repeated vomiting prevent the extreme meteorism characteristic of intestinal paralysis.

6. Localized tenderness and percussion-dulness. When present these signs are of exceeding great importance, since they denote the position of the strangulated bowel.

Exceptionally a tumor may be felt formed by the congested gut or the matting together of the intestinal coils.

The urine is diminished in quantity and may be suppressed. In acute strangulation it commonly contains albumin, and it is stated that this is of diagnostic value.

In this connection the history is of great importance.

Other congenital deformities would suggest the possibility of Meckel's diverticulum as a cause.

A preceding typhlitis, pelvic peritonitis, or severe abdominal traumatism would respectively assign an adherent vermiform appendix, peritoneal bands, or rents in the omentum or mesentery as the causative agents in the production of the symptoms.

The age of the patient should also be considered, since this form of obstruction usually occurs between the twentieth and fortieth year.

The sudden onset of violent, persistent pain, subnormal temperature and frequent pulse, the obstinate, absolute constipation, the persistent, repeated vomiting, becoming fecal, and the rapid course of the disease, all point to internal strangulation.

Auscultation of the abdomen is at times of value, a sound compared to the click of the water-hammer being heard most distinctly at the point of obstruction.

Palpation and percussion should not be omitted, as thereby the seat of obstruction has been distinctly located.

Volvulus is the most frequent form of intestinal obstruction after intussusception and that due to strangulation. Vomiting occurs, but is not so constant a symptom as in those forms first named. Thus it occurred in from 8 per cent. in Brinton's statistics to 2.5 per cent.

in those of Treves, and 4 per cent. in Martin's and the author's. It is nearly always seen in men in middle life. The vomiting is rarely fecal, is very slight in many cases, and sometimes does not appear at all.

Vomiting, on the other hand, is quite commonly seen in cases of *obstruction from impaction* or obstruction from foreign bodies. This underlying cause of the emesis can be diagnosed by the history of a foreign body having been swallowed, of attacks of hepatic colic, or, where a gallstone ulcerates through into the bowel, of some local peritonitis about the region of the liver. A history can commonly be elicited of sharp, colicky pain, of partial obstruction, and of vomiting. The distention is slight, the amount of systemic shock far less than in other forms of obstruction, and the duration of the attack somewhat longer than usually obtains in this class of affections. The symptoms of obstruction are frequently only partial, the vomiting being moderate in amount and not stercoraceous, the constipation not being absolute.

Except in the case of enteroliths and very large foreign bodies a tumor can rarely be felt.

It is often impossible to diagnose this form of obstruction from that depending upon a narrowing of the lumen of the bowel, such as is produced by cancer or stricture. The previous history is always of great importance. The presence of indican rather than albumin in the urine, the comparative mildness of the attack, the moderate meteorism, and the slow course of the disease, all help to exclude internal strangulation or volvulus. It is, however, mainly upon the history that the diagnosis will be founded.

Vomiting, loss of appetite, thirst, cough, hectic fever, and sweats, with the development of marked cachexia, sometimes occur from *obstruction of the rectum.*

When vomiting arises from *peritonitis* it is often one of the earliest symptoms of the malady. It is almost always present and is often a very severe symptom, and is associated with, or replaced by, a constant retching, which adds to the exhaustion of the patient. At first it may only follow the swallowing of food, but often it occurs without such a cause, and after the stomach is emptied of its ordinary contents glairy, watery mucus is expelled, which is often of a distinct greenish tint. The great tenderness of the belly in acute peritonitis, the moderate fever, the rapid pulse, the anxious face,

[1] For the test for indican, see chapter on the Urine.

and the cold skin as collapse approaches, all render the diagnosis easy; but it is to be remembered that the distention of the belly by an overfilled bladder or pregnant uterus may mislead the physician into thinking that peritonitis is present because of the swelling, the pain, and the vomiting. Vomiting is not a severe symptom of appendicitis unless the peritoneum has become involved in the inflammatory process, although it may occur once or twice when the pain in the appendix is most severe. The localization of the symptoms in the neighborhood of the appendix makes the diagnosis possible. (See chapter on Abdomen.) When vomiting occurs in typhoid fever it is usually a symptom of bad feeding or imperfect digestion, and is rarely of grave importance except under two conditions. The first of these is when it occurs as a result and symptom of intestinal perforation, an accident commonly seen late in the disease; and, second, when it takes place as an obstinate and exhausting symptom after the fever has practically passed by, from unknown causes, probably reflex in character. The symptoms of perforation other than vomiting can be found in the chapter on the Abdomen and Abdominal Viscera.

Vomiting as a symptom of *cholera* is accompanied by serous diarrhœa of a profuse character, by the development of collapse, cramps in the muscles, anuria, and great circulatory failure. It must be separated from the vomiting due to cholera morbus or severe indigestion, antimonial poisoning, and arsenical poisoning. Cholera morbus is to be separated from cholera, first, by the absence of the comma-bacillus in the stools; second, by the fact that there is a history of exposure to cold or damp; third, by the absence of an epidemic; and, fourth, by the milder manifestations. No one can be skilful enough to separate symptoms of poisoning by antimony from that due to cholera, for the symptoms are identical in every way. Nothing but the history of the ingestion of the poison and the discovery of antimony in the secretions can prove the case to be one of antimonial poisoning, particularly if an epidemic of cholera is present.

In *arsenical poisoning* the association of vomiting with bloody stools separates the symptoms from those of cholera.

Vomiting is a very severe and early symptom of *cholera infantum* (see chapters on "Abdomen" and "Bowels and Feces"), and it occurs in attacks of *true dysentery* as a common symptom, when its underlying cause is readily discovered. (See Abdomen.) The dis-

eases of the stomach causing vomiting are cancer, ulcer, gastritis, catarrh (acute and chronic), true gastritis, and dilatation.

The vomiting of *gastric cancer* at first consists in the expulsion from the stomach of its contents—mixed particles of food, mucus, water, and sometimes bile. The vomit may be tasteless or sour from fermentation, and may have an offensive odor from similar causes. Often it contains blood either in bright-red streaks or as a brownish-red fluid, or in similarly colored clots, which may be brown when they have been in the stomach for some time. Often the exuded blood, changed by mixture with the stomach-contents, looks like coffee-grounds, producing "coffee-ground vomit." This coffee-ground vomit is not pathognomonic of gastric cancer, but is very characteristic of this disease. Microscopically the vomited materials are seen to consist of particles of food, yeast-cells, cocci, and broken-down blood-corpuscles. (For the other symptoms of gastric cancer, see chapter on the Abdomen.)

Coffee-ground vomit is also sometimes seen in cases of *locomotor ataxia* following a gastric crisis.

Vomiting due to *gastric ulcer* is preceded by pain, and is generally brought on by taking food, and so occurs soon after eating. The food is, therefore, only slightly digested, and evidences of fermentation are absent to a great extent. If blood is present, it is nearly always bright red and in considerable quantity, and indicates that a hemorrhage has recently taken place from the surface of an ulcer. Very large hemorrhages into the stomach may cause vomiting by irritating and distending this viscus. The history of vomiting after eating, the presence of blood in the vomit, the pain in the stomach, the age of the patient (generally twenty to thirty years), the sex (generally female), and the hyperchloric acidity, combined with the other symptoms (see chapter on Abdomen), complete the diagnostic array of facts.

There are, however, other causes of vomiting of blood or hæmatemesis than gastric ulcer and cancer. Thus it occurs from obstruction to the portal circulation from *hepatic cirrhosis*, and from growths and splenic affections which result in varicosity of the bloodvessels of the stomach. Hæmatemesis also follows severe blows, kicks, and other injuries to the epigastrium. Sometimes it takes place in cases of heart disease in which there has resulted hepatic engorgement with secondary gastric congestion, and it may be developed in small degree by any form of violent vomiting which strains the stomach,

particularly if an irritant substance has already destroyed the mucous membrane. Again, hæmatemesis is seen in scurvy, typhus, yellow fever, and acute yellow atrophy of the liver, as a result of breaking down or destruction of the coats of the vessels. Sometimes it is seen in cases of dengue, in influenza of the epidemic type, and in relapsing fever. Hæmatemesis may also occur in purpura hemorrhagica, in hæmophilia, and as a result of vicarious menstruation. In malarial fever of a severe character the dark-colored vomit is generally due to bile, but it may be due to exuded blood. Such a case is reported by Boon as occurring in a child.

Care should always be taken that the physician is not misled by the vomiting of swallowed blood into a diagnosis of gastric hemorrhage from any of the causes just named. It may enter into the stomach from the pharynx, as after epistaxis, or blood may be swallowed by a malingerer. Hæmatemesis is to be separated from hæmoptysis by the fact that in the latter there are physical signs in the lungs, the sputum is frothy and bloody, there is absence of retching or vomiting-movements, and the blood is bright red in hæmoptysis oftener than in hæmatemesis.

In order to determine that the discolored vomit of any case is due to blood a microscopical examination for the corpuscles must be made, and if these are greatly altered a chemical test may be used. (See further in this chapter.)

The development of vomiting with sudden pain in the abdomen, resembling colic, which fails to yield to ordinary remedies, and is associated with sources for an embolism, should lead the physician to a consideration of possible *embolism in the superior mesenteric artery*. This condition must not be confounded with the vomiting of *acute pancreatitis*, in which colicky pain in the epigastrium, deeply seated and extending to the right shoulder and back (see Hepatitis, in this chapter), and great restlessness, præcordial distress, dyspnœa, and faintness are present. The matters vomited are greenish, clear, and viscid, and the efforts at vomiting increase the pain. There is no jaundice, and death soon comes to the relief of the patient.

As an early diagnosis of acute pancreatitis may permit surgical interference with possible recovery of the patient, the diagnosis is important. The mistake commonly made is to consider the case one of intestinal obstruction.

Under the name of melæna neonatorum there is a condition of hæmatemesis occurring in children within the first few days or weeks of life. This condition has been thought by Leube to be due to gastric

and duodenal ulcers, and his views are indorsed by Buhl and Huhn, Spiegelberg, Binz, and Landau. In one of the latter's cases thrombosis of the umbilical vein was present, and it has been thought that when expansion of the chest takes place in the newborn child small clots may escape from this vessel through the ductus arteriosus into the descending aorta and gastric arteries, and thus cause an ulcer of the stomach by embolism.

Vomiting of a peculiar character is always present in *phosphorus-poisoning*. The symptoms associated with ingestion of the poison may not come on for some hours. At the end of that time the peculiar taste of phosphorus may be noticed in the mouth, the breath is heavily laden with its odor, and burning pain in the œsophagus, stomach, and abdomen ensues. Vomiting and purging now assert themselves, and both the matters vomited and passed from the bowels may be luminous in the dark, owing to the presence of phosphorus. The vomiting is at first made up of food, then mucus, then bile, then perhaps blood. All the symptoms of a mild gastro-enteritis may develop, but it is to be noted that constipation of an obstinate type may replace the purging. Very soon the liver increases in size, and gives rise to general hypochondriac pain and tenderness, as well as local swelling. At the end of twenty-four hours, or perhaps after the second day, a cessation in the symptoms occurs, and, if the physician be not on his guard, this will lead him to a hopeful prognosis. In the course of a few hours jaundice begins in the conjunctiva, and then extends over the entire body. With the onset of jaundice the vomiting and pain return with renewed vigor. The matters vomited are often the color of "coffee-grounds," due to exuded and altered blood. The bowels are absolutely confined, or the few hard masses passed are white and clay-like, because of the absence of biliary coloring-matter. There is no bile in the vomit in this stage, because the hepatic ducts have been closed by the inflammation set up in the liver. After this nervous symptoms ensue. Muscular twitchings, headache, vertigo, wild delirium, erotic convulsions, and finally unconsciousness and death occur. Sometimes the convulsions occur just before dissolution. Even if the patient survive the acute stage, he generally dies of the changes produced in his vital organs, which consist in widespread fatty degeneration, even in the acute stages. Atrophy of the liver, destruction of the gastric tubules, pancreatic involvement, and kidney degenerations aid in producing the ultimately fatal result.

Vomiting also arises from *neuroses of the stomach* in several forms. Thus it is frequently seen in hysteria, in neurasthenia, pregnancy, and sometimes occurs in the form of what Gee has called "cyclical vomiting." It also comes on in association with gastric crises in locomotor ataxia. The vomiting of *hysteria* is generally characterized by its persistent character, often lasting for months, and yet the patient often maintains her normal weight to a surprising degree, either because the food which is taken is only vomited in small part or because she surreptitiously obtains food when her attendants do not know it, which she retains. It is generally associated with so many of the hysterical stigmata as to be readily diagnosed. The vomiting of *neurasthenia* is seen in both sexes, and is particularly apt to follow any nervous or muscular exertion. Thus in one case of the writer's even short railroad journeys taken by an overworked man produced attacks of spinal tenderness with vomiting, which lasted several days. The vomiting of *pregnancy* is usually a morning vomiting, though it may persist all through the day. It has no particular diagnostic features in itself, save that there are rarely any signs of gastric indigestion. The presence of pregnancy, of course, makes the diagnosis clear; but in such cases, if the pregnancy is advanced, the physician should always examine the urine, since the ordinary vomiting of pregnancy is a symptom of the first few months, while that occurring later on may indicate grave renal complications. (See Uræmia, under chapter on Coma and in chapter on Convulsions.)

The *cyclical vomiting* already mentioned is generally seen in children, and is of rare occurrence. It is characterized by attacks of vomiting recurring after intervals of uncertain length, during which the patient may seem entirely well. The attack may last from a few hours to a few days. There are often pain in the epigastrium and constipation. Sometimes retching is the main symptom. It is generally seen in neurotic patients.

Vomiting of a nervous type is a common complication of *exophthalmic goitre*, and when it occurs sometimes develops into a dangerously severe symptom, owing to its constancy, violence, and resistance to treatment. Oftentimes serous diarrhœa is associated with it, and these two associated symptoms should not mislead the physician into a diagnosis of cholera morbus nor of jaundice, for icterus often comes on.

Ménière's disease is associated with vomiting, the contents of the stomach being expelled after the attack of vertigo and tinnitus aurium.

The *affections of the liver* which result in vomiting are chiefly catarrhal and obstructive jaundice, hepatitis, and pylephlebitis. The rapid development of jaundice, hepatic tenderness, and swelling, or a history of violent hepatic pain (colic), renders the diagnosis possible in the case of jaundice. (See chapter on Skin.) Hepatitis—that is, hepatic abscess—is often accompanied by or produces vomiting which is apt to be very obstinate. The swelling of the liver, the tenderness in the hypochondrium on palpation, the pain in the hepatic region, often referred to the neighborhood of the right shoulder, and the febrile movement, which is intermittent, sweeping up to 104° or 105°, then down to normal, are the chief characteristic symptoms. (See chapter on Abdomen.) Vomiting accompanied by a similar train of symptoms also occurs in cases suffering from pylephlebitis.

Violent vomiting associated with great pain in the loin, radiating down into the testicle or inside of the thigh, indicates the presence of a *renal calculus*, either in the pelvis of the kidney or in the ureter.

Hæmoglobinæmia is sometimes accompanied by vomiting. The attacks are paroxysmal, and are often ushered in by persistent yawning, with pain in the limbs, headache, nausea, and vomiting, followed by moderate fever, which is preceded or accompanied by a chill. Pain may be felt in the liver, but, more pathognomonic than all, the urine is soon found to be dark, brownish-red, or even black, owing to the presence in it of hæmoglobin.

Vomiting is a frequent coincident symptom of headache, because in many cases the headache depends for its existence upon a disordered stomach or disordered bowels; but it also appears as a characteristic symptom of a condition in which the stomach is primarily not at fault, namely, in *migraine* or *hemicrania*, in which, in addition to violent pain in the head on one side, we have hemianopsia, scotomata, and sometimes great pallor or flushing of the face. (See Pain.) In *acute yellow atrophy of the liver* vomiting is a constant symptom, with jaundice, violent headache, and finally convulsions and subcutaneous ecchymoses. (See chapter on Convulsions.)

When vomiting occurs in *yellow fever*, the presence of an epidemic, the suffusion of the eyes, the headache, the black character of the vomit, the slow pulse, scanty urine, and prostration, all point to the real cause of the symptom. In *Addison's disease* vomiting is often present, and may be so severe as to prove beyond control. The peculiar pigmentation of the skin (see Skin), the mental inactivity,

headache, vertigo, and anæmia without loss of weight in many cases, all aid in the diagnosis.

Vomiting is a frequent symptom in some cases of *phthisis*, particularly if laryngeal tuberculosis is present. It also occurs as a result of swallowing the sputum instead of expectorating it, and very commonly excessive cough produces vomiting, especially if the cough follows closely after a meal.

Closely associated with the vomiting due to these causes is that occurring in cases of *pulmonary gangrene.*

Vomiting often takes place in children suffering from *whooping-cough* at the close of the paroxysm, and is due to the spasmodic movements of the chest and diaphragm.

Finally, it is not to be forgotten that vomiting often ushers in any one of the *eruptive diseases*, such as the fevers, syphilis in its early secondary stages, and erysipelas.

Under the name of merycismus cases of *voluntary regurgitation* of food have been reported. The patients have been nervous or hysterical persons.

The Vomit.

Aside from the diagnostic significance of the act of vomiting, the physician should remember that the vomit itself may give him information as to the condition of his patient.

Under the head of gastric dilatation we have spoken of the significance of vomiting large amounts of liquid and undigested food, so that it is not necessary to speak of this point here; but it is well to remember that small amounts of vomited material often possess considerable diagnostic importance. Thus, in the case of drunkards it is by no means rare for the patient to vomit in the morning small amounts of watery and sour material, and in the severe retching of cerebral disease or uræmia very little material is gotten rid of. In cases in which small quantities of exceedingly sour, clear liquid are vomited, we often find that the attack is due to migraine or nervous headache. If watery liquid and mucus are vomited, there is probably gastric catarrh. The vomiting of bile is usually only seen when repeated retching has drawn this secretion up into the stomach. The liquid may be either golden yellow or greenish in hue. Somewhat like this vomit is that seen in peritonitis, in which disease grass-green material is often expelled. Similar material is also

vomited in cases of intestinal obstruction before stercoraceous vomiting comes on

The significance of bloody vomit has already been described. It only remains to name the test for hæmin, which, if present, shows that the color of the ejecta is really due to blood. Some of the vomited material is filtered, and a little of the filtrate is evaporated on a watch-glass. This dried material is now scraped off the glass, mixed with a trace of finely powdered common salt, and placed on a glass slide. The powder is now covered by a cover-glass and one or two drops of glacial acetic acid allowed to flow under the cover-glass. This is then dried by exposing it to gentle heat, and after the drying is absolute a drop or two of pure water is touched to the edge of the cover-glass. Very minute crystals of hæmin are now seen with the aid of the microscope (see Fig. 188).

FIG. 188.

Hæmin crystals.

The vomit of uræmia is often ammoniacal from the decomposed urea in it, and that of intestinal obstruction is fecal in odor for obvious reasons. If odorous poisons have been taken, it smells of the poison; and if there be phosphorus present, the vomit not only smells of it, but in addition is luminous in the dark.

CHAPTER VI.

COUGH AND EXPECTORATION.

The varieties of and indications of cough—The causes of cough—The sputum—Its pathological significance.

THE significance of cough as a symptom is very important, and, though it may arise from many causes, in the majority of instances it points to trouble in the chest, in the trachea or the larynx, in the pharynx or in the nose. Rarely it is a purely nervous trick, and equally rarely it arises from irritation in the stomach ("stomach-cough," so called). A cough is said to be dry and hacking when it fails to bring up into the throat or mouth any secretion, or when it is short and sharp. Often such a cough is paroxysmal; in other cases it consists in single but fairly frequently repeated, short, and forcible expiratory efforts, as if the patient was trying to clear his throat. A loose cough is nearly always paroxysmal; that is, it occurs in "spells," and at nearly every paroxysm results in the raising of some mucus. The first variety of cough is that seen in the early stages of phthisis pulmonalis, acute bronchitis, or pneumonia, before any exudation has taken place; in the early part of a paroxysm of asthma; in the early portion of an attack of whooping-cough, and when the cough arises from irritation in the upper air-passages, whether this be due to the inhalation of dust or the presence of some growth, as a laryngeal papilloma. The loose variety of cough is seen in the later stages of acute bronchitis, pneumonia, asthma, whooping-cough, and in cases of emphysema with bronchiectasis, and in the stage of pulmonary tuberculosis associated with the breaking down of lung-tissue, the formation of cavity, and the development of bronchitis with it, and in gangrene of the lung. There are two peculiar forms of cough to be mentioned, namely, the so-called barking, brassy, laryngeal cough, which we hear most typically in false or spasmodic croup, and the cough of whooping-cough, which is, as its name implies, the most typical which we meet with. Suddenly the child begins to give a series of quick, sharp coughs, which become more and more rapid until the chest is emptied of air. In the early stages of the disease this is all that occurs, and unimpeded inspiration ensues; but later the cough no

sooner ceases from exhaustion of the lungs of air than with the attempt of deep inspiration the glottis closes spasmodically, and the air is sucked in through the chink with a whooping sound. The flushed or cyanotic face of the child, associated with these paroxysmal attacks, renders the diagnosis easy.

There is nothing distinctive in the cough of early stages of pulmonary inflammation, whether it be bronchial or vesicular, although, if the bronchitis be very intense or the pulmonary inflammation also affect the pleura, the cough may be partly smothered or suppressed by the patient, who endeavors to control or stop it in order to escape the pain it causes. To this end he sits or lies in bed, endeavors to fix the muscles of his chest so that they will not respond to the reflex cough impulse, and shuts his lips to hold his breath in, although very often the reflex irritation overcomes his will-power and the cough bursts through his compressed lips with an expression of pain. Such a cough is always indicative of pain.

In all forms of dry cough there is now and again a small plug of mucus expelled from some part of the respiratory mucous membrane. Such coughs possess no value to the patient, being merely a sign of reflex irritation; but a loose cough, unless it is very excessive, is of the greatest possible use to the patient, for it is an effort on the part of nature to rid the lungs of abnormal exudations or secretions. For this reason this symptom is not to be removed completely in cases of resolving pneumonia, pulmonary tuberculosis, or bronchiectasis with excessive secretion, since, if drugs are given which stop the cough, the lungs are speedily filled with the secretion; and in the case of tuberculosis or gangrene or muco-purulent bronchitis septic absorption with systemic infection results. Similar good results are reached by the cough heard in cases of pulmonary abscess, and when an empyema has broken into a bronchial tube. When the patient complains of chronic cough, which is worse in, or confined entirely to, the morning hours, and tells us that the cough finally causes the discharge of much secretion, and that this is followed by freedom from cough for many hours, the case may be one of tuberculosis with cavity; pulmonary abscess; empyema, which has ruptured into a bronchus; or sacculated bronchiectasis. Such coughs come on in paroxysms whenever the lung must be relieved, and the length of paroxysm depends upon the looseness of the secretion and its situation in the lung. Thus, if the secretion be in the larger bronchial tubes, it is easily expelled; whereas if it be in smaller

bronchioles, or in the vesicles, or at the bottom of a cavity, great and frequently repeated effort will be required before the liquid can be raised into the mouth for expectoration.

The presence of an obstinate cough due to bronchitis, which resists all ordinary treatment, should lead the physician strongly to suspect that one of two ailments is present, namely, undiscovered tuberculosis, or, if there is puffiness under the eyes, Bright's disease.

Cough brought on by changing the position of the patient often arises because of the alteration in position of a pleural effusion. Violent and constant cough often also comes on during aspiration of the chest for pleural effusion.

The cough of acute laryngitis may be quite severe, and occurs in short, sharp barks of a harsh or brassy character (like spasmodic cough), which is so typical as to be called a laryngeal cough. The association with this cough of partial or complete loss of voice and pain in the larynx, with a history of exposure to cold and dust, or the excessive use of the larynx in speech or singing, renders the diagnosis clear even if the laryngoscope is not used to discover congestion and inflammation of the laryngeal mucous membrane. In the false croup of children, which is always associated with laryngeal irritation, the barking, ringing cough is so characteristic as to render a diagnosis possible as soon as the sound is heard, and with it there is dyspnœa due to obstruction to breathing.

The cough of laryngeal phthisis is not so typically brassy and ringing as that of acute laryngitis, but the presence of pain in the larynx, hoarseness, and persistent laryngeal dryness and difficulty should lead to a search for tuberculosis by the laryngoscope, and an examination of the chest for physical signs of trouble in the lungs and of the sputum for tubercle bacilli.

Sometimes cough of a laryngeal character is due to an aneurism pressing upon the larynx. In other cases the cough depends not upon the pressure of an aneurism, but upon the pressure produced by carcinoma of the œsophagus, by a mediastinal tumor, or is due to the inhalation of irritant dusts or vapors. This cough, laryngeal or bronchial, is often present in girls who work in carpet factories, in the air of which there are immense quantities of fine particles of wool. Again, it is seen in knife-grinders, needle-workers, coal-miners, and in workers in arsenical and lead pigments.

Sometimes in paralysis of the pharyngeal muscles (glosso-labio-

pharyngeal paralysis) cough is produced by the slow passage of food, which may in fact enter the larynx.

A night or evening cough is very commonly seen in cases of respiratory catarrh or more grave disease. It often is absent all day, only to return in the evening in cases of laryngitis and in phthisis; and, in those cases in which it follows getting into bed, it is due to chilling of the skin by the cold sheets, which results in congestion of the inflamed mucous membrane.

Quite frequently children suffering from chronically enlarged tonsils suffer from cough on going to sleep, especially if the uvula is relaxed or elongated. The cause of this cough is that in the relaxation of sleep the tonsils touch one another or tickle the uvula. As soon as the child wakes muscular contraction separates the approximating surfaces and the cough soon ceases.

Sometimes in hysteria a peculiar barking cough is produced by pinching the skin of the neck or elsewhere in the body, and it may also come on in paroxysms independent of such causes in neurotic children of both sexes about the time of puberty.

In regard to coughs in general, it can be said that in the absence of the early stages of acute inflammation of the respiratory apparatus a dry, hacking cough is either nervous or is due to reflex irritation in the ear or stomach, or to some hyperæsthetic spot in the nasal, pharyngeal, or laryngeal mucous membrane; whereas a loose cough may arise both in adults and children from congestion and engorgement of the lingual tonsil. Such a cough is frequent, dry, and paroxysmal, and seems to arise from a sticking or pricking sensation in the throat; whereas a loose and prolonged cough depends upon causes of greater gravity further down in the respiratory organs. Generally, if the stomach is at fault, a low grade of pharyngitis will be found. The physician should also remember that valvular disease of the heart, by the secondary changes which it causes in the lungs, may produce cough, and Sansom states in his book that cough was present in 45 per cent. of the cases of heart disease taken by him as illustrative.

There is another variety of cough seen in persons who have paralysis of the diaphragm. The cough under these circumstances is produced for the purpose of expelling air from the chest, and is seen in persons with paralysis of the diaphragm from the removal of large ovarian tumors, which have pressed upon it and stretched it and the chest-walls to which it is attached, so that when the pressure is

removed the muscle is too slack to contract. It is also seen in injuries to the phrenic nerve and to the spinal cord.

The cessation of cough in advanced phthisis, suffocative bronchitis, or the bronchorrhœa with bronchiectasis of old persons, or in severe pneumonia, indicates exhaustion, collapse, or approaching unconsciousness, and is a bad sign.

The Expectoration.

A careful examination of the materials expectorated by the patient, or, in other words, of the sputum, is of the utmost importance in all cases of disease of the respiratory tract, whether the abnormal process be primary or secondary. The methods which we resort to in examining sputum are macroscopic, or those of the naked-eye, and microscopic. Of these the microscopic are by far the most important. Sputum varies greatly in its general character on ordinary examination, sometimes being very fluid and even watery in consistency, and sometimes thick or tenacious. In some instances it is clear and glairy-looking, resembling somewhat partly beaten white of egg; in others it is yellow and opaque. Placed on a clean linen cloth the sputum may evaporate to almost nothing, or leave a heavy muco-purulent deposit after all moisture is gone.

The naked-eye appearances of sputum are, however, quite characteristic in several conditions. Thus, in the later stages of acute bronchitis the sputum is apt to be thick and yellowish and to contain lumps of half-inspissated mucus. In croupous pneumonia it is rusty in color, is peculiarly free from watery ingredients, and is gelatinous to such an extent that it adheres to the spit-cup, so that when this vessel is well filled its contents do not readily fall out even when the cup is tipped upside down. Later on it may be less adhesive after resolution is well advanced. If the pneumonia is chronic, the sputum has such a dark color that it is often called "prune-juice sputum." A third variety is that seen in pulmonary hemorrhage or hæmoptysis. In this condition, after having, perhaps for a short time, a salty taste in the mouth, the patient suddenly brings up, with or without much preceding cough, a gush of nearly pure blood or blood freely mixed with ordinary sputum. The blood is bright red, not dark or prune-juice in appearance, and the liquid is frothy, while the cough, which is always present after the hemorrhage has occurred, is suppressed and resisted by the patient, who

fears further bleeding. This hæmoptysis may be caused, first, by pulmonary tuberculosis; second, by valvular cardiac disease, generally involving the mitral valves; third, by aortic aneurism; fourth, in persons suffering from severe purpura; fifth, from persons suffering from hæmophilia; sixth, from vicarious menstruation; and, seventh, in rare cases of hemorrhagic smallpox.

Bloody sputum must be separated from bloody vomit due to gastric hemorrhage from ulcer or cancer (see Vomiting). This can be done by the cough, by the frothy character of the expectoration, by the presence of physical signs in the lungs, and by the history of pulmonary disease. It may, however, be confused with slight hemorrhage from a dilated and ruptured vessel on the posterior pharyngeal wall, in which case, after a little coughing, there may be expelled on a handkerchief a little blood-tinged saliva. Examination of the throat will usually reveal the source of the blood or other vessels dilated, but still intact. For a number of days after an attack of hæmoptysis there may be expelled in the sputum dark clots of blood. So-called "currant-jelly" clots are expelled by coughing in many cases of malignant growths of the lungs.

Other causes of blood-streaked sputum are aortic aneurism with leakage by oozing into a bronchus or the trachea; valvular disease of the heart, causing pulmonary engorgement; and particularly in children do we see streaks of blood in the sputum if there be present pulmonary gangrene. Care should always be taken to discover whether the materials spat up are really tinged with blood, for they may be colored by some dye-stuff or the blood of some animal for purposes of deception.

Finally, it is well to remember that a reddish-brown or brick-dust looking sputum is sometimes coughed up in cases of hepatic abscess communicating with the lung; and the sudden expectoration of a brownish, purulent-looking sputum by a person who has been a sufferer from dysentery should cause the physician to examine the sputum for the amœba coli, in order to discover if the case is one of pulmonary abscess secondary to amœbic dysentery. Symptoms of hepatic abscess may also be present. This has been called "anchovy-sauce" sputum.

In addition to the sputum already described we sometimes see a peculiar semi-liquid sputum in cases of pulmonary phthisis, in which the sputum promptly separates into two layers on standing, the lower one being light and flocculent, unless there is a well-marked bronchial

tedious and complicated process, and for the discovery of Charcot-Leyden crystals and fibrinous coagula the centrifuge is a much better apparatus. (See chapters on Urine and Blood.) The tubes used for the precipitation of the sputum are 50 mm. long, with $2\frac{1}{2}$ mm. lumen, and are fitted into the hæmatocrit frame.

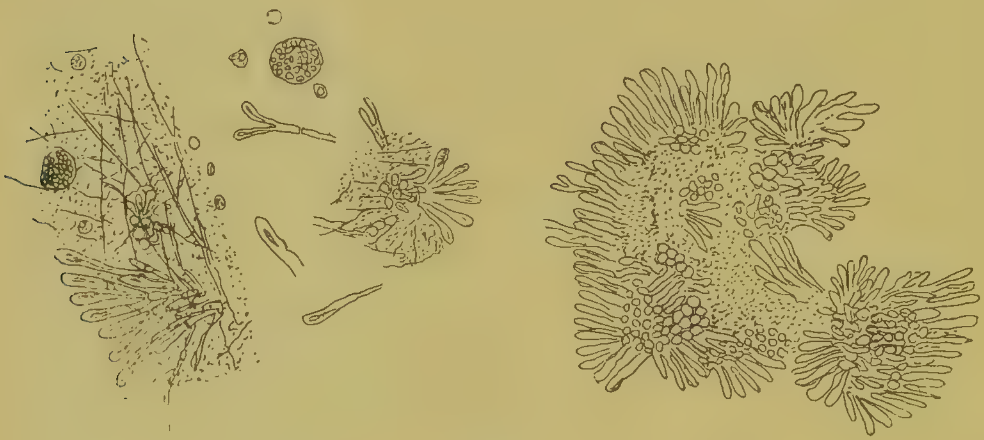
A small quantity of sputum is placed in a clean porcelain dish and actively stirred for a few minutes with a glass rod until it becomes thin and free from lumps and apparently homogeneous. Without any dilution whatever the sputum is drawn into the sputum-tubes by means of a medicine-dropper connected with a small rubber tube. The two precipitating tubes, filled with sputum in this way, are placed in the hæmatocrit frame and revolved for at least three minutes, making about fifteen thousand revolutions. The solid portions of the sputum collect in the distal extremity of the tubes. A small portion of the sediment is placed on a slide and examined microscopically for elastic fibres, Charcot-Leyden crystals, etc. The sediment from the second tube can be stained for tubercle bacilli or other micro-organism as desired.

When the centrifuge is not used and a small particle of sputum is placed upon a glass slide under a cover-glass and gently pressed there will be seen, if the sputum be mucus or muco-purulent, threads of mucus and mucous corpuscles with white blood-corpuscles, which are particularly numerous if the sputum be purulent. These latter are granular, fatty, and sometimes pigmented by soot or other substances which have been inhaled. Epithelial cells derived from the respiratory passages are also found in large numbers, often broken down and fissured, granular, and generally a nucleus can be distinguished in their centre. Of far more importance than these, however, are particles of elastic fibre or elastic threads, which, if present, show positively that a breaking-down process is going on in the lung, or more rarely in the bronchial tubes. These are usually seen in the sputum of advanced or rapidly progressing tuberculosis of the lung and in that of abscess and gangrene of the lung. If there is doubt as to their presence because they are sparse, we obtain them by the following process : Boil equal parts of the suspected sputum and a 10 per cent. solution of caustic potash, and dilute the jelly-like mass which results with water. After this has stood for twenty-four hours the elastic fibres may be found in the sediment as swollen threads, for which, however, small particles of food which may come from the mouth may be mistaken.

The appearance of fine, needle-like crystals of fatty (margaric) acid, which may be bent like a bent needle and often grouped in bunches, may indicate pulmonary gangrene or purulent bronchitis. They are found chiefly in the plugs or lumps which the patient expels in his sputum, but they possess no indicativeness of the pulmonary changes just named if follicular tonsillitis, either acute or chronic, is present, since the plugs derived from the follicles of the tonsils also contain similar crystals. Again, they are of no diagnostic value if found in stale, muco-purulent sputum, as they may form in this after it has been expectorated. The peculiar crystals called Charcot-Leyden crystals have already been described.

There are four remaining objects to be seen in sputum of diseased persons, all of which are of great diagnostic importance when found.

Fig. 190.



Actinomyces.

The first of these is very rare, namely, the eggs of the distoma pulmonum, which are found in the sputum. This parasite sometimes produces hæmoptysis without any physical signs of pulmonary change, and is rarely if ever seen in this country, but is common in Japan, Corea, Formosa, and in China. The second is the evidence of echinococcus infection by the appearance of portions of the cysts or of the hooks of the scolices when the cyst bursts into a bronchus from the lung or adjacent structures. Such cases are rare in this country. The third condition, which is also very rare when involving the lung, is actinomycosis, in which infection we find radiated fungi or actinomyces in the sputum. This fungus appears as a number of club-shaped projections attached to a heterogeneous mass of irregular-looking material. (Fig. 190.)

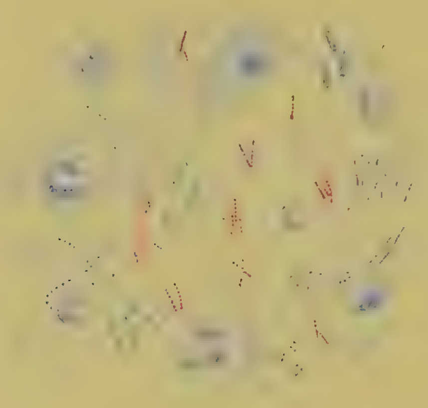
The fourth and most important of all these finds in sputum is the tubercle bacillus, both from the point of diagnosis, prognosis, and treatment, and because the disease tuberculosis is so widely distributed in every class, in every part of the country, and is so constantly prevalent. The discovery of these bacilli in a sample of sputum, which has not been exposed to the entrance of bacilli after it has been expectorated, is a positive sign of tuberculous infection unless there be tuberculous disease of the upper air-passages or mouth. While their presence gives positive evidence, their absence in a given sample of sputum is not negative evidence of an absolute character, for that particular specimen may be free from bacilli or they may have escaped the staining or the eye of the examiner.

The method for examining sputum for tubercle bacilli is as follows : The specimen which is brought to the physician is poured into a shallow glass vessel having a blackened bottom, or into a glass saucer placed on a piece of blackened paper. If this sputum is now closely examined, it will be found to contain small, yellowish masses, one of which should be picked up by means of a spoon or platinum needle, freeing it as much as possible from the mucus surrounding it. A very small part of this mass is now placed on a cover-glass and well distributed over its surface by means of a needle or teasers, or it is spread by placing another cover-glass over the first and pressing them together with a to-and-fro movement. The quantity of sputum used must not be large enough to extend over the edges of the glass. This having been done, the glasses are now separated by a gliding movement, and the thin film of sputum covering each one is allowed to dry by exposure to the air, after which, being held by forceps, it is passed through the flame of an alcohol lamp to fix the coating. Care must be taken that too much heat is not used. Some carbol-fuchsin stain[1] is now placed in a watch-glass and the cover-glasses are immersed in it. As soon as this is done the cover-glass is held over a flame until steam begins to rise from it, when it is withdrawn, then heated again until this process has been repeated several times. The cover-glass is now washed in water, and the film covering it will be found to have an evenly distributed red color. The glass is next placed for a moment in a 25 per cent. solution of nitric or sulphuric acid in water and gently moved about to decolorize the deposit on the surface of the glass. As

[1] Carbol-fuchsin stain, or Ziehl's solution, is made by dissolving 1 gramme of fuchsin in 10 c.c. of alcohol, and adding 100 c.c. of a 5 per cent. solution of carbolic acid.

a result the albumin and cells on the glass are decolorized, but the bacilli are not. The glass is next washed in dilute alcohol (60 per cent.) to remove any free fuchsin. Should the decolorization be imperfect, so that the film still has a red color, it must be still further decolorized by returning the glass to the acid solution and then washed again in the dilute alcohol and water. The cover-glass is now placed in a solution of methylene-blue (saturated watery solution). The glass is then finally washed in water and afterward examined under the microscope, or dried and mounted in Canada balsam for permanent preservation. The tubercle bacilli are distinguished by the fact that they retain the red color from the fuchsin solution, while other bacteria, having been decolorized by the acid solution, take the contrast-stain and appear blue.

FIG. 191.



Tuberculous sputum stained by Gabbett's method. Tubercle bacilli seen as red rods; all else is stained blue. (ABBOTT.)

Another method (Gabbett's modification of Fraenkel's) is perhaps more useful than that just given. It consists in staining, as already directed, with the carbol-fuchsin solution, and then placing the cover-glass in a second solution, which contains the acid for decolorizing and the contrast-stain. This latter solution is composed of 20 parts of nitric acid, 30 parts of alcohol, 50 parts of water, and sufficient methylene-blue to make a saturated solution, equalling above one or two parts in a hundred. The cover-glasses are left in this solution for a couple of minutes, and then washed in water. When placed under the microscope the tubercle bacilli will appear as red rods in strong contrast to the blue background.

CHAPTER VII.

PAIN.[1]

The kinds of pain—The significance of its locality—Colic.

It is manifest that it will be impossible for the author in this volume to enumerate all kinds of pain, both as to the situation, degree, and character. He can only mention those forms which possess considerable diagnostic importance. It should always be remembered that pain is the sign adopted by nature to notify the individual of some abnormal condition in his body, and in many instances pain is only developed when the attempt is made to move a part which from its condition had much better be allowed to rest.

Pain is generally described as darting or stabbing in character, when it occurs in a single or repeated paroxysm; as throbbing or pulsating, when it rises and falls in severity with the pulse-beat; as dull and aching, when it resembles the feeling associated with a bruise. Sometimes stabbing or darting pains are called lancinating, and often the patient will state that the pain is tearing and rending in character. All pain is associated with direct and indirect irritation of nervous matter, and, if the nerve or nervous centres connected with a part be destroyed organically or in function, we have a condition called anæsthesia. (See chapter on Skin.)

Almost invariably darting or stabbing pain is associated closely with actual disease of nervous tissue, which may be primary or caused by the pressure or irritation of a growth or some foreign body. Such pains are seen in neuralgias due to inflammation of the sheath of a nerve or its surroundings as it passes through a bony foramen; in neuralgia due to meningeal thickening; in the agonizing lightning or tearing pains of locomotor ataxia; of pressure upon the spinal nerves by spinal disease or in that caused by fractured bones. Again, we often meet with violent pain as the result of true neuritis, whether it be produced by infection, by injury, or by poisoning.

Throbbing pain is nearly always associated with the presence of congestion or local swelling in the part where the pain originates, and arises from the fact that the peripheral nerves are subjected to

[1] See also chapter on Headache.

pressure, which is increased with each additional beat of the heart. Dull, aching pain is often produced by slow inflammatory or pathological processes in organs not well endowed with sensory nerves.

There are two forms of pain yet to be considered which are peculiar in their character, namely, that due to a blow or injury to the testicle and ovary and that which occurs in cases of inflammation or morbid growth affecting bony tissues, particularly in the long bones.

There is another point in connection with the study of pain as a symptom of disease, namely, that pain is often referred to a point far away from the source of the symptom. Thus, the child with hip-disease complains of pain in the knee or in the ankle; the one with dorsal caries of pain in the intercostal nerves anteriorly; and a stone in the kidney may cause violent pain in the penis or testicle.

The physician should always remember that the degree of pain must be determined in part by the expression of the face and movements of the body, for often these features of a case will show that the pain described so vividly in words is much exaggerated. The general systemic signs of pain are a tense pulse, if the pain be recent in onset and acute; a somewhat accelerated respiration unless the pleuræ or lungs are involved, when it may be retarded; dilatation of the pupil; more or less sweating, particularly on the forehead; faintness; and sometimes the passage of clear, limpid urine if the pain be abdominal.

The first kind of pain which will be discussed will be the darting or paroxysmal pains in that division of pains which may be called the *neuralgias.* These depend upon one of three causes, and, though they may occur in any nerve of the body, are most commonly seen in the nerves of the head; or in nervous women, in the nerves of the pelvic organs and external genitals. The three causes are generally debility with anæmia, reflex irritation, and irritation of the nerve by poisons or by the presence of growths.

Violent neuralgia of the head is commonly seen in overworked or overdanced young women, who lack sufficient sleep and fresh air and who are anæmic. It also arises from the reflex irritation of a decayed tooth, or from an inflamed or overstrained eye, or of a diseased ear, so that an examination of any one of these parts may reveal the cause of an obstinate neuralgic pain. Similarly we see cases of neuralgia, particularly of the supraorbital nerve, which are due to chronic poisoning by one of the metallic poisons, such as lead and arsenic, and also as a result of malarial infection (brow ague). If

the neuralgic pain be due to neuritis, it will not only be typical of neuralgia, but along the track of the nerve marked tenderness will be developed on pressure, and often an eruption will appear on the skin, as a herpes zoster. Pure neuralgia, on the other hand, is often relieved by pressure upon the nerve involved.

When the fifth cranial nerve is affected by neuralgia we find that if the upper branch is involved the pain is felt in the forehead, the eyebrow, and the eyeball, the conjunctiva often becoming injected. If the pain be in the upper lip, the posterior nares, and the cheek, the infraorbital or second branch is affected; while if the pain is in the lower jaw and chin, the third division of the fifth nerve is involved.

A peculiar form of neuralgic pain coming on in attacks or paroxysms of great severity is *migraine* or *megrim*, in which the pain is usually confined to one side of the head, is associated with great tenderness of the scalp, and preceded in many cases by disorders of vision, such as hemianopsia or dimness of visual perception. Associated with this pain at its zenith we frequently see vomiting and retching, faintness with sweating localized to the pain-area or diffused, and great facial pallor. Pressure by the fingers upon the painful area often gives no more pain or even partial relief, but a light touch will often cause marked increase in the pain. Rarely a somewhat similar condition to migraine, which is not unilateral but bilateral, is found in connection with rheumatism of the scalp. As both migraine and this last-named condition arise from the rheumatic diathesis, care in making a differential diagnosis is necessary. The pain of migraine is, however, unilateral, more severe, more transitory, and associated with the symptoms named, whereas in the rheumatic head-pain the history of rheumatic tendencies of a marked character, the diffuse pain, the increased soreness on exposure to cold or changes in the weather, aid in separating it from migraine. When syphilis or injury causes a periostitis of the skull, violent pain of a neuralgic character may be present, particularly at night; but the local symptoms are manifest, and when compared with the history make the diagnosis possible.

It is also necessary to separate the headache of *cerebral tumor* or cerebral abscess from neuralgia of the head. The pain of such a cerebral condition is constant; the headache is sometimes worse at night, sometimes in the daytime, and greatly increased by physical or mental effort. The danger of confusing the pain of neuralgia with

that due to tumor is great unless the physician remembers that the constant pain of tumor may vary from slight headache to sharp paroxysms of pain. The occurrence of convulsions points strongly to tumor if associated with headache of this character, and, finally, the presence of tumor as a cause of headache and not ordinary neuralgia is decided upon by evidences of optic neuritis, vomiting, vertigo, and, finally, the development of focal symptoms of localized paralysis. (See chapter on Headache and Vomiting, Convulsions, and Spasms.)

The most common seat for neuralgic pain in the head, other than in the brow, is the occipital region, the posterior branch of the second cervical nerve or great occipital being the one most affected. As this nerve supplies all the occipital region and the posterior part of the parietal regions, all these areas may be involved in the painful manifestations, and all these parts may be tender to the touch. Brushing the hair may be impossible, because of the pain produced by the brush touching the scalp. Occipital neuralgia is oftentimes bilateral. It may simply arise from cold or injury ; but, if persistent and severe, caries of the cervical vertebra should be sought for as a possible cause.

Pain of a neuralgic or darting character in the neighborhood of the heart is found as a result of several causes, as a rule in the following order of frequency : 1. Pain, with palpitation in the præcordium from the accumulation of flatus in the transverse colon just as it turns to descend. Many patients who come to the physician complaining of heart disease only suffer from this condition, due to fermentation. Again, the pain due to gastralgia, or, as it has been called, cardialgia, may be referred to the heart by the patient. 2. To intercostal neuralgia due to debility. In these cases a tender spot will often be found, one in the præcordium, another in the outer edge of the scapula, and a third on the vertebral column. These are sometimes called the "spots of Valleix." In other cases it will be due to spinal trouble, anæmia, or the tight lacing of corsets. 3. To pseudo-angina. 4. To true angina pectoris. 5. To locomotor ataxia.

Pain of a character somewhat resembling true angina pectoris is also sometimes met with in patients who have that rare disease, *acute aortitis*. The pain is constant under the sternum, but it has terrible exacerbations, and a sensation of rending of the retro-sternal tissues. Death may occur in an attack. It is seen chiefly in gouty patients and in syphilitics. Very rarely it is seen in patients who have suffered from malarial poisoning.

Pain is felt much more commonly in disease of the aortic orifice than in lesions of the mitral orifice of the heart.

Pseudo-angina occurs most commonly in anæmic, nervous girls, or young men whose vessels are normal, but who have hysterical tendencies. *True angina* occurs in those of middle age or advanced life or in young persons whose vessels are affected by syphilis. In the false form the sensation is as if the heart would burst. In the real form it feels as if the heart was squeezed tight in a vise. In this form, too, the bloodvessels will usually be found hard and corded, atheromatous, and the blood-pressure high. The additional diagnostic points in favor of true angina pectoris are that the principal seat of pain is somewhat to the left of the lower and middle sternum, from which spot it may extend to the axilla and back and turn off to the occiput or extend down the arms to the hands, where a sensation of coldness may be felt. Sometimes even the abdominal organs and testicles seem to be affected. The patient is motionless, the face anxious and covered with a cold sweat, and respiration is shallow. The disease is usually seen in persons over forty years of age. The thoracic pain of locomotor ataxia is rarely in the præcordium, but commonly in the axilla, and it rarely radiates down the arm. The other symptoms of tabes dorsalis should be sought for in all doubtful cases. (See chapter on Legs and Feet.)

Very severe pain, paroxysmal or constant, felt in the chest may also be due to *aortic aneurism*, and, if so, will be found associated with pain shooting down the arm on the left side, dilatation of the pupil, unilateral sweating of the face and neck, and the physical signs described in the article on the Chest.

Neuralgia of the *pelvic viscera* in women is frequently seen as the result of functional or organic disease. It may be ovarian, when it is very apt to occur with greatest severity half-way between the menstrual epochs or just before them. Sometimes the neuralgia may be present in the labia majora or in the perineum. It usually occurs simply as a sudden darting pain, which does not last and, indeed, rarely continues more than a moment, although there is usually associated with it more or less constant uterine or ovarian tenderness. Care should be taken that these pains are not thought to be due to cancer or other severe organic lesion. Pain in the sacral region is often an indication of uterine or rectal disease. If higher up the back, it is often due to myalgia or lumbago; and

lumbago, if not due to rheumatic tendencies, is often due to the colon being loaded with feces.

If the patient is a child, pain in the back should cause us to suspect *spinal caries, rickets,* or *scurvy.* If the former, any jar will greatly increase the pain ; but if the child be placed over the knees, face downward, and the knees separated so that intervertebral pressure is removed, the pain disappears. Such a child if told to jump down from a stool will not obey, but will take care to slide off gradually and gently on to the floor.

When a patient suffers from violent pain, increased by motion, extending from the sciatic notch in the buttock down the posterior part of the thigh, even to the ankle or heel, the pain signifies an attack of *sciatic neuralgia* in an adult, or if it occurs in a child gives grave reason for suspecting *hip-disease.* If it is not sciatic neuralgia, it is due to sciatic neuritis, or, rarely, to a growth in the pelvis pressing upon the nerve before it emerges from the pelvis. The pain is fairly constant, generally worse at night, and becomes agonizing at times, even if the patient remains absolutely quiet and does not move the limb. The following points will, when pressed on, increase the pain if it be neuritis : The point of exit from the nerve from the pelvis, the lower part of the sacrum, the head of the fibula, and behind the malleolus on the outside of the ankle. If these points are found, combined with a history of exposure to cold, injury to the nerve, rheumatic tendencies, and a persistency and tendency to return, the diagnosis of sciatica is clear. If the pain be due to sciatic neuritis, there may be found wasting in the muscles supplied by the nerve, and some anæsthesia of the skin, and herpetic eruptions may appear on the skin along the course of the nerves. There will also be a history of a long duration, and the leg will be apt to feel numb and tense from effusions into the sheath of the nerve. (See chapters on Skin and Feet and Legs.) When the pain is a pure neuralgia, which is rare, it will not be increased by moving the limb, there is little or no tenderness on pressure on the nerve-trunk, and the patient often has neuralgia of other nerves.

Sciatica is much more common in men than in women, and far more usual in middle or advanced age than in the young.

Double sciatic pain should arouse suspicion of *locomotor ataxia,* of malignant growth pressing on the spinal cord or on both nerves in the pelvis, or the presence of lumbar abscess.

When there is hysterical, painful joint at the knee or hip in a

woman, care is necessary to discover that the pain is over the entire leg rather than in the course of the nerve. Care must also be taken that rheumatism of the muscles of the thigh be not taken for sciatica. This can be separated from sciatica by the diffuse character of the pain and tenderness and by the fact that in the rheumatic condition the slightest muscular movement causes pain all over the thigh. Sometimes a *malignant growth of the femur* may produce symptoms of sciatica, and the writer not long since had under his care a case of osteosarcoma of the thigh bone which had been treated for sciatica for several months.

Finally, *renal calculus* may cause violent pain to pass down the leg. (See below.)

It should also be remembered that malingerers, particularly soldiers desiring to shirk duty, often pretend to have sciatica.

Neuralgic pain in the back and abdominal parietes very closely resembling, if severe, renal or hepatic colic is sometimes seen in *gastralgia*, and the paroxysms may be very sudden in onset. In other instances the pain is in the epigastrium or hypogastrium, and is associated with so much tenderness on light pressure as to impress the careless with the belief that a gastric ulcer or cancer is the cause. Neuralgic spots can generally be isolated in such persons if the skin is carefully tested for its degrees of sensation, and will be found to exist near where the nerves are given off from the spine or over the spine of the ilium. There are other causes of these lightning abdominal pains than simple functional neuralgia, so-called, and renal and hepatic colic, which should never be forgotten in the diagnosis of a case, namely, *locomotor ataxia* manifested in gastric crises, lead-poisoning shown by a neuralgia due to a neuritis, and disease of the vertebræ causing pressure on the nerve-trunks.

Further than this, we are to remember that *cancer* and *ulcer* of the *stomach* often cause exceedingly severe, painful attacks; that abdominal aneurism may cause severe pain by pressure on nerves; and that uterine and periuterine disease also cause, reflexly, epigastric pains.

Abdominal Colic. By colic we mean a sudden, griping pain in the belly. It is met with chiefly in cases of stone in the ureter or gall-duct, but it is often due to irritating food, to wind, and to chronic lead-poisoning, to fecal impaction, intestinal obstruction, intestinal perforation, and ulcer of the bowels.

Reference has already been made to the pain of *renal* and *hepatic colic*. The characteristic symptoms of these conditions are as follows: In renal colic the patient is suddenly seized with violent pain in the kidney on one side, which passes down to the groin and even to the end of the penis. It is paroxysmal in character, and so excessively severe that it often produces sweating, vomiting, and even fainting. The condition is seen much more frequently in men than in women. The pain often suddenly subsides, leaving only a sense of soreness and tenderness in its track. The urine may be partly suppressed and bloody if the stone injures the ureter to any great extent.

In hepatic colic the patient often, after some days of wretchedness and "biliousness," is seized by sudden and violent pain in the right hypochondrium, which is paroxysmal in character and causes quite as profound general symptoms of disorder as does the renal colic. Jaundice very commonly ensues in such cases with more or less rapidity, and fever of an irregular type is more often seen than in the renal form. The pains just described are so severe and characteristic in their distribution that they cannot well be confused with those of intestinal indigestion, in which condition we have a history of the ingestion of bad food, a state of more or less flatulent distention of the entire belly, and, it may be, diarrhœa. Sometimes violent belly pain is due to aneurism. If the pain be due to chronic lead-poisoning, it centres about the umbilicus, and is of a twisting, knotty character, "as if the bowels were being twisted around a stick." There is a history of exposure to lead in many cases, and a blue-line on the gums can often be found. If due to fecal impaction, we will have a history of a continued tendency to constipation, with dry, hard stools, and a lump of hardened feces may perhaps be felt through the belly-wall. In perforation of the bowel the patient speedily becomes collapsed, and suffers severe pain. This accident generally occurs in persons convalescing from typhoid fever. If due to intestinal obstruction, the pain has no characteristic seat in any part of the abdomen, as a rule; but the general symptoms of this condition will be found present in the case. (See chapter on Abdomen or that on Vomiting.)

The pain of colic due to flatulence can be separated from that of peritonitis by the fact that pressure gives comfort in the first instance and is unbearable in the latter disease.

Pain in the abdomen of the darting neuralgic type, other than that

due to gallstones, renal calculi, ordinary gastralgia, lead-poisoning, enteralgia, or malignant growth, may be due to *locomotor ataxia.* This should never be forgotten, and the fact that the patient is full-grown, complains of the most violent pain in the belly, and has no other abdominal signs, should make us search him for the other signs of tabes dorsalis. Generally, these attacks will be of a tearing, rending character, are beyond description in severity, and leave the man in a condition of nervous wreck after them. Sometimes the pain is in the stomach, sometimes in the bladder.

Neuralgia of the toe and foot is not a very rare condition, and is sometimes called "Morton's painful toe," or *metatarsal neuralgia.* Severe pain at the base of the fourth toe comes on suddenly, and may radiate up the anterior aspect of the leg. Sometimes it is only dull, but at others it is so sharp and excruciating as to cause the patient to scream. It is separated from gout by the absence of any signs of inflammation in the part, by the fact that the big toe is not affected, and by the age and history of the patient. At times the base of the second toe is affected. Such a case will usually indicate that the patient has worn an ill-fitting boot.

Finally, in connection with this class of cases there should not be forgotten two others, namely, those in which, idiopathically or otherwise, growths form on nerves and cause pain ; and, second, cases in which the arm or leg, having been amputated, a *neuroma* or catching of the end of the nerve in the scar causes violent pain in the lost part, according to the patient's sensation, because the perceptive centres have been trained to regard pain-impulses coming along this nerve as from its peripheral end. Thus a man whose leg may have been amputated years before will complain of severe pain in the amputated foot although he knows it is off.

The pain of inflammation is often very severe and throbbing in character ; but, if the nerves be affected in the area involved, it may be darting in its nature. We have already discussed pain due to inflammation in the tissues of the head and in the nerves. We now have to consider the indications of pain due to inflammation in the chest and abdomen.

Severe pain of a darting character felt in the chest, not due to angina, is nearly always an indication of one of four things : 1. Intercostal neuralgia, already named. 2. Pleuritis, with or without pneumonia. 3. Pericarditis, if it is felt in the præcordium. 4. A

morbid growth in the chest, particularly a mediastinal tumor or enlarged bronchial glands.

Both *intercostal neuralgia* and *pleurisy* are associated with severe pain, increased by taking a deep breath, the pain occurring sometimes with inspiration and sometimes with expiration. They are to be separated one from another by the presence of cough, fever, and of a friction-sound in pleuritis, and by the fact that the entire side is more or less tender to the touch in this state. When the pain is constant and lasts for a long time, it may be due to a low-grade pleuritis, resulting from pulmonary tuberculosis, particularly of the apex of the lung, the morbid process affecting the pleura. *Pericarditis* is frequently caused by pneumonia, and is painful.

Pain felt at the right of the left scapula or between the shoulders is often due to gastric ulcer or dyspepsia.

The pain of mediastinal growth is due to pressure on nerve-trunks, and the diagnosis may be very difficult unless bulging and dulness on percussion are present.

Generally diffused pain of a constant severe character felt all over the abdomen or localized at first to some particular spot, and greatly increased by pressure, should lead the physician to examine the case for a possible *peritonitis.* Nothing else causes such violent, diffuse pain unless it be acute enteritis. The drawn-up legs, the anxious face, the drawn upper lip, quick pulse, exquisitely tender abdominal surface, the thirst, the moderate fever, and the rapid onset of collapse in fatal cases render the diagnosis easy.

Circumscribed abdominal pain of a constant character and generally of less severity than that just described, may be due to dysmenorrhœa, to an abdominal tumor (see Abdomen), to an ovaritis, to cystitis, hepatitis, ulcer of the stomach or bowel, typhlitis, perityphlitis, appendicitis, and cholangitis. It is also seen in a very violent form in acute pancreatitis, which may give rise, with the other symptoms, to a diagnosis of peritonitis. In *dysmenorrhœa* the pain is sometimes so severe as to render the patient almost insane, but it differs from that of inflammation in that it is paroxysmal and that there is no real tenderness on pressure; and, again, the patient does not lie still, but tosses from side to side in the bed. The pain of *tumor* is usually produced by pressure on a nerve, and is increased by palpation in some cases, as is also that of ovaritis. In *cystitis* the pain is deep in the pelvis, radiating upward, and is associated with tenderness, vesical spasm, and tenesmus. In *hepa-*

titis the pain is distinctly in the hypochondrium, although if the condition be suppurative it may be well diffused. The pain of ulcer of the stomach is not only gastric, but is often associated with a tender or painful spot at the angle of the scapula. In *typhlitis*, perityphlitis, and appendicitis it is chiefly in the right groin, and in cholangitis in the hepatic area anteriorly. Care should be taken that the pain in this region is not taken for hepatitis, when it is in reality due to a *subphrenic abscess*. In *pancreatitis* the pain is sudden in onset, violent, and often felt chiefly in the left upper zone of the abdomen. The belly is distended, nausea and vomiting are present, and fever may be present also; delirium may come on, and death generally speedily ensues.

The pain of *fissure of the anus* is not at all proportionate to the lesion producing it. This pain may be atrocious and agonizing, and often is produced by a movement of the bowels, after which it lasts for some hours.

(For abdominal pain due to conditions associated with movements of the bowels, see chapter on Bowels and Feces.)

Pain in the back is often very severe in the early stages of small-pox, of epidemic influenza, and in lumbago. One of the most misleading forms of pain of a severe character, involving the entire body, with fever, delirium, and a variable skin eruption and swelling of the joints, may in the early stage be thought to be smallpox or rheumatic fever, when in reality it is due to *dengue* or breakbone fever.

CHAPTER VIII.

TENDON-REFLEXES AND MUSCLE-TONE.

The knee-jerk and ankle-clonus—The arm-jerk—The significance of decreased and increased reflexes.

We have already had occasion, particularly in those chapters devoted to the Legs and Feet and Hand and Arm, to speak of what are called the reflexes or "muscle-jerks." There is much discussion as to whether the muscular contractions produced by tapping the tendon attached to a muscle are the result of a reflex action, in which the spinal cord is directly involved, or whether it depends upon muscle irritability or tone. It is not necessary for purposes of diagnosis to enter into a discussion of this character, because the facts in our possession prove conclusively that variations in these muscle-jerks are of great diagnostic importance in diseases of the nervous system, whether they be true reflexes or not. The knee-jerk, or, as it has been called, the patellar reflex, is the diagnostic sign most frequently sought in studying nervous diseases associated with lesions in the spinal cord, because it is most easily developed.

This knee-jerk is best developed by directing the patient to sit in an ordinary chair and to cross the legs, the foot which touches the floor being kept near the rounds of the chair rather than well forward, and the upper leg being allowed to swing perfectly freely over the under knee. By means of the finger-tips, the side of the hand, or a small rubber hammer, a blow is now struck upon the tendon of the quadriceps extensor midway between the patella and the insertion of the tendon below the knee, and, if the man is healthy, the hanging leg and foot are at once thrown forward; that is, the knee-jerk is developed.

If there be produced by the patient at the moment of the tap on his tendon a strong muscular effort, as by sudden clinching of the hands, this movement of the leg is exaggerated, or, to use the common term, the knee-jerk is "re-enforced."

The production of pain or a sensory stimulation will also increase the knee-jerk, as will also the stirring of the emotions as by lively music. On the other hand, enervating weather, loss of sleep, hunger,

or anything which decreases general systemic and muscular tone decreases the knee-jerk.

The second most important test of the muscular tone is the test for what is called "ankle-clonus." If a healthy person while sitting rests the weight of the leg on the ball of the foot, the leg is very apt, in a short time, to begin to tremble, and finally to develop clonic movements. In disease these movements often develop as soon as the foot is placed in this position, and are at once developed if, while the leg is so placed, a sharp blow is struck above the knee or sudden pressure is made on the leg. In other cases the clonus is tested for by grasping the ankle with one hand and the toes with the other and bending the foot up toward the knee with a rather forcible push, which will stretch the muscles of the calf of the leg.

The biceps tendon is tested by placing the arm in semi-extension and tapping the tendon, when the forearm is still further flexed. In this connection mention should also be made of the cremasteric reflex, which is developed when the skin of the inside of the thigh is tickled, the cremaster muscle drawing up the testicle on that side. It is most marked in boys.

Having learned how to test for these muscle-jerks, we now turn to a consideration of what they mean when abnormally increased or absent.

A loss of knee-jerk is not characteristic of any disease unless this loss is associated with other symptoms, which only need the discovery of this symptom to confirm the diagnosis. The nervous conditions in which we find the reflexes decreased or lost, taking the patellar reflex as a type, are locomotor ataxia ; peripheral neuritis ; poliomyelitis, acute or chronic ; transverse myelitis, if the disease involves the reflex arc ; Friedreich's ataxia ; diphtheritic paralysis ; apoplexy, immediately after the shock ; Landry's paralysis ; spinal meningitis; spinal injuries, immediately after accident; epilepsy, immediately after an attack ; and chorea. We also find a total loss of reflexes in advanced diabetes mellitus and sometimes in diabetes insipidus.

By far the most common cause of the loss of knee-jerk is locomotor ataxia, but any lesion involving the posterior columns of the cord or the posterior nerve-roots in the second, third, or fourth lumbar segment will produce the same results. Therefore, loss of knee-jerk is symptomatic of transverse myelitis of this region as well as of ataxia. Again, if the motor tract of the cord at these levels

are diseased, the knee-jerk is lost, as, for example, in acute and chronic poliomyelitis or myelitis involving the motor part of the reflex arc; and, finally, peripheral neuritis, which blocks the pathway from the periphery to the cord, and from the cord to the muscles, also causes loss of knee-jerk.

If the cause of loss of knee-jerk be locomotor ataxia, we will probably find in addition to this symptom some difficulty in walking, particularly if the eyes are shut; a lack of steadiness if the feet are placed together when the patient stands with his eyes shut; Argyll-Robertson pupils or a reaction to accommodation, but not to light; attacks of severe pain in the body or limbs; and, it may be, laryngeal crises or spasms and atrophy of the optic nerve.

If the cause of loss of knee-jerk be neuritis, we will find tenderness on pressure along the nerve-trunks, diminished muscular tone and some wasting; an absence of any disturbance of the bladder and no Argyll-Robertson pupil, laryngeal or other crises, nor optic atrophy.

Again, if the cause be acute poliomyelitis, there will be a history of sudden onset with fever, the limbs will be relaxed and flabby, the muscles will rapidly waste and become very feeble or paralyzed, and there will be no sensory symptoms whatever. The patient will usually be a child if the disease is acute. If the loss be due to a transverse myelitis of the second, third, and fourth lumbar segments, the symptoms of paraplegia, paræsthesia, and anæsthesia, with atrophy of the muscles and loss of control of the bladder and rectum, will be present, and a girdle-sensation may be marked.

In Friedreich's ataxia the history of heredity, the nystagmus, the early age of the patient, the absence of pupillary symptoms, the ataxic gait, and the loss of reflexes, are the facts which go to form our basis for a diagnosis. In the remaining diseases named the history of the case points to the cause of the loss of the knee-jerk very clearly.

The conditions in which we find the knee-jerk *increased* are apoplexy soon after the attack; disseminated sclerosis; cerebral palsy of childhood; paretic dementia (not constant); primary lateral sclerosis; amyotrophic lateral sclerosis; ataxic paraplegia; hysterical paraplegia; transverse myelitis if the lesion is above the reflex arc; epilepsy some minutes after the attack; unilateral lesions of the cord on the paralyzed side; injuries to the spinal cord, after recovery from first shock; pressure on spinal cord above the reflex arc; heredi-

tary cerebellar ataxia; sciatica; tetanus; rheumatoid arthritis; and neurasthenia.

The history of sudden paralysis and unconsciousness in a case of apoplexy with stertorous breathing, followed by loss of the knee-jerk, and then its return in an exaggerated manner, make the diagnosis clear unless the attack be one of the apoplectiform attacks of disseminated sclerosis, in which case there will be present a history of the intention-tremor, nystagmus, and the syllabic speech, so that though the knee-jerk is exaggerated in both diseases the diagnosis can be readily made. In the cerebral palsy of childhood, the age of the patient, the contractures and gait, with the history, decide the diagnosis. In lateral sclerosis the spastic rigidity, excessive exaggeration of the knee-jerks, absence of sensory disturbances, and ocular symptoms, all render the diagnosis possible. Similar exaggeration is also seen in amyotrophic lateral sclerosis, in which disease there is wasting of the muscles, particularly of the hand. In both these ailments the exaggeration of the knee-jerk is due to disease of the lateral pyramidal tracts, which block the inhibitory fibres from the higher centres. For similar reasons we find exaggerated knee-jerk in ataxic paraplegia.

In hysterical paraplegia the age and sex of the patient, the peculiar facies, the areas of anæsthesia and hyperæsthesia, and the peculiar gait point to the diagnosis.

The increased knee-jerk in cases of transverse myelitis occurs when the lesion is situated at such a point in the cord that the lateral tracts are cut off and the reflex arc is preserved.

In neurasthenia the knee-jerks are exaggerated, but are easily exhausted.

Leaving the knee-jerk as a type of a reflex, we find that the skin-reflexes are often lost in cases of apoplexy when the deep reflexes are exaggerated. The table on following page from Taylor's *Index of Medicine* shows the area of skin reflexes very well.

In glosso-labio-pharyngeal paralysis the reflexes of the tongue and throat are lost and those of the face sometimes increased; in progressive muscular atrophy the reflexes of the arms are lost, while those of the legs are preserved, and in tubercular meningitis the reflexes are apt to be more marked on one side than on the other:

In athetosis the reflexes are increased in the affected part.

Ankle-clonus is found most marked in lateral sclerosis, in disseminated sclerosis, and in amyotrophic lateral sclerosis. A false clonus is sometimes seen in hysteria.

Reflex.	Point of stimulation.	Situation of centre.	Significance.
1. Plantar,	Irritating skin of soles.	Extreme end of cord.	Usual in health.
2. Gluteal,	Irritating skin of buttocks.	Origin of 4th and 5th lumbar nerves.	Rare in health.
3. Cremasteric,	Irritating skin of inner side of thighs.	Origin of 1st and 2d lumbar nerves.	Usual in health; best marked in boys on account of the newly formed cremaster.
4. Abdominal,	Irritating skin of abdomen in line of nipples.	Origin of 8th to 12th dorsal nerves.	Frequently absent.
5. Epigastric,	Irritating skin of chest in 5th and 6th spaces.	Origin of 4th to 6th dorsal nerves.	May be absent in health.
6. Erector spinæ,	Irritating skin from scapula to crest of ilium.	Origin of all the dorsal nerves.	Rare in health; frequent in wasting disease.
7. Interscapular,	Irritating skin between scapulæ.	Origin of 6th cervical to 3d dorsal.	Rare in health.
8. Palmar,	Palms of hands.	Cervical bulb.	Only in infants.
9. Cranial:			
Conjunctival,	Sclerotic, or inner surface of eyelid.	Medulla.	Absent in disease of 5th nerve only.
Iris (to light),	Pupil.	Anterior portion of oculomotor nucleus.	Absent in disease only.
Palate,	Soft palate and uvula.	Medulla.	Absent in disease only.
Nasal (sneezing),	Naso-respiratory passages.	Medulla.	Absent in disease only.

CHAPTER IX.

SPEECH.

The changes in the speech and voice—Their significance—Aphasia—Apraxia—Alexia—Paraphasia.

THE character of the speech and the tones of the voice often convey a considerable amount of diagnostic information to the physician. While in many diseases no marked alterations from the normal manner of speech are present, in others marked changes take place. Thus, in acute laryngitis due to exposure to cold or irritant vapors the patient has a *whispering* voice. In persons suffering from pulmonary tuberculosis the development of hoarseness and whispering, or labored speech tells us only too well of the fact that the grave and distressing complication called laryngeal tuberculosis has arisen, and that the progress of the case will be more rapid toward the fatal result. Again, the sudden onset of whispering voice or complete aphonia, occurring in a young girl whose facies is hysterical, should always arouse a suspicion of hysteria, while if the signs of this condition are absent and the patient has none of the signs of tuberculosis, we should examine the larynx for a papillomatous growth. Again, if *hoarseness* or a whispering voice is manifested by a male of adult years, who is also suffering from dyspnœa, unilateral flushing or sweating of the face and neck, and unequal radial pulses, we should suspect aortic aneurism or a mediastinal tumor which is pressing on his recurrent laryngeal nerve. When a child speaks with a *nasal twang* or indistinctly we suspect the presence of adenoid vegetations, and will probably find that he or she suffers from mouth-breathing; and stuttering or stammering may also be due to this cause.

A *feeble, hesitating speech* is often a sign of exhausting disease, and a short and quick but feebly spoken sentence generally indicates that the patient is suffering from some cardiac or pulmonary complaint, which renders his breath short, so that he hurries through his sentence in order to be able to breathe freely again. Thus, in cases of pneumonia or of pulmonary œdema this hurried speech is a very constant sign.

Again, in cases of typhoid fever, when the tongue is dry and immobile from accumulated sordes, a mumbling character of the speech is present, even if the brain is entirely clear, and in severe stomatitis the same quality of the voice may be present.

It is in connection with the disorders of the nervous system, however, that the most typical alterations of the voice occur. Let us suppose that a patient in middle life or in more advanced years develops a *slow, scanning speech,* with intention-tremors (see chapter on Hand), nystagmus, and more or less muscular weakness. In all probability he is a sufferer from insular, or, as it is otherwise called, disseminated sclerosis. When he speaks each syllable is sharply accentuated and slowly pronounced. The only other conditions in which a slow, scanning speech is of great diagnostic importance is in that rare disease, Freidreich's ataxia ; but the fact that this disease begins in childhood, that several members of the family are apt to be affected, that there are ataxic symptoms and early talipes equinus, renders it easy to separate this affection from insular sclerosis. (See Paraplegia, in chapter on Feet and Legs.)

A *hesitating, halting speech* associated with Argyll-Robertson pupils, unequal pupils, delusions of grandeur, and tremor of the tongue, which last symptom may be so marked as to cause the speech to be indistinct and blurred, is indicative of paretic dementia.

If an incoherent speech develops in a child who is not suffering from an acute illness causing delirium, there will usually be found in association with this symptom the nervous twitchings of chorea, for speech-disturbances occur in about one-third of the patients suffering from this disease.

A very *indistinct speech of a mumbling character,* great difficulty being experienced in the pronouncing of dental and lingual sounds, and perhaps associated with feebleness of the voice, if the larynx is involved, is seen in cases of glosso-labio-pharyngeal paralysis. If the cause of the defective speech be this disease, there will be found, as associated symptoms, wasting of the tongue, lingual tremors, some dribbling of saliva from the mouth, and immobility of the lips, the face about the mouth being expressionless.

Somewhat similar symptoms due to paralysis of the lips, with escape of the tongue and pharynx, at least for a long time after labial paralysis develops, is sometimes seen in advanced cases of amyotrophic lateral sclerosis ; and a still more close resemblance

may be produced by the so-called "pseudo-bulbar paralysis," the lesion of which is in the motor cortex of the brain on both sides, in the lower part of the ascending frontal convolution. Rarely the latter is only a unilateral disease.

A rather *shrill, piping voice,* the sentence being begun with hesitation and then hurried to an end in rapid volley of words, is sometimes seen in paralysis agitans.

By far the most interesting speech-defect is that called *aphasia.* It is divided into motor aphasia and sensory aphasia.

Before studying these conditions we must discuss the nervous mechanism of speech. When a child learns to talk it performs a purely imitative act. Its auditory nerve conveys the sound to its perceptive centres, and from here an impulse is sent to its motor speech-centres, and these again send impulses to the inferior speech-nuclei in the medulla oblongata, which in turn move the muscles of speech. Simultaneously the child learns the words and stores them in memory-centres for sounds, and also stores in memory-centres "motor memories," which tell him how to repeat the muscular movements a second time. Again, when he learns to recognize objects and call them by name he must use "visual memory" centres. These centres are all best developed in the left hemisphere of the brain in right-handed persons and in the right half of the brain in left-handed persons.

If a person suffers from pure aphasia, he simply loses the memory of how to say certain words, and the lesion is in the third left frontal (Broca's) convolution. He can read to himself, because he has not forgotten the meaning of the words, and for this reason he understands what is said to him, and may be able to repeat a word immediately after you have said it by a purely imitative process. Generally we find with aphasia a condition called *agraphia* in which the patient cannot write voluntarily, but can copy perfectly. In the great majority of cases of aphasia, however, the patient is paralyzed in his right hand, so that the symptoms of agraphia cannot be demonstrated. Under the name of *paraphasia* we sometimes meet with a condition in which the patient can speak quite freely, but transposes words or interpolates useless words to such an extent that what he says is unintelligible.

In another condition closely connected with aphasia we have a state in which the patient can spell out words from a page set before him, but he cannot read, because the words convey no idea to him.

This is called *alexia* or "word-blindness." Again, he may forget the use or significance of certain objects, such as a knife or fork; this is called *apraxia*. Still further, words when spoken to the patient in his native language may be heard perfectly, and yet understood no more than if in some unheard-of language. This is called "word-deafness."

If the patient has simple aphasia, he has a lesion in the third frontal convolution in its posterior part. If he has word-blindness or alexia, the lesion is in the angular gyrus, extending back into the occipital convolution. If he has apraxia or the loss of memory of objects, the lesion is in the same area as in alexia; and if "word-deafness" is present, the lesion is in the posterior part of the first temporal and upper part of the second temporal convolution. As the various symptoms of aphasia in all its forms are closely associated with those of focal lesions of the brain, resulting, for example, from hemorrhage or embolism, the reader should read the chapter on Hemiplegia in this connection.

The following plan of testing a patient, devised by Eskridge from a shorter one of M. Allen Starr, may be followed with advantage:

1. The power to recognize objects seen, heard, felt, tasted, smelt, and their uses.

2. The power to recall the spoken names of objects seen, heard, felt, tasted, and smelt.

3. The power to understand sounds other than speech.

4. The power to understand speech and music.

5. The power to call to mind objects named and point them out at request.

6. If word-deaf, can he recognize his own name when it is spoken?

7. The power to recognize a word spelled aloud.

8. The power to call up mentally the sound of a note, figure, letter, or word.

The examination thus far will test the various sensory areas, but more especially the auditory and the association tracts between the different sensory areas connected with speech.

9. The power to recognize letters, figures, notes, and colors seen.

10. The power to understand printed and written words seen.

11. The power to read printing, writing, and music aloud and inaudibly, and to understand what he reads.

12. The power to recall objects, the names of which are seen.

13. The power to write voluntarily.

14. The power to write at dictation.

15. The power to copy, and the manner of copying, printing, and writing.

16. The power to write the name of objects seen, heard, felt, tasted, and smelt.

17. The power to read aloud and inaudibly, and to understand what has been written.

18. The power to write his name and the ability to read it when written by himself and by another person, or when it is printed.

19. The power to recognize a letter by tracing it with the index-finger or with a pencil, the movements being guided by another.

20. The power to call up mentally the appearance of an object, a figure, a note, letter, or word, when word-blind.

These additional tests will aid in determining the condition of the visual word-memories in the angular gyrus, and the connection between this area and the surrounding sensory and motor areas:

21. The power to speak voluntarily, and, if impaired or lost, the character of the defect.

22. The power to repeat words after another.

23. Does the patient recognize his mistakes in speaking and writing, and can he correct them?

24. Can the patient think in speech (propositionize)?

25. Is there any special difficulty in the use of nouns, verbs, or other parts of speech?

26. The power to understand pantomime or gesture expression.

27. The power to employ intelligently gesture in expression.

28. The power to read figures and to calculate.

29. The power to count both money and in numbers.

30. The power to play a game of cards or other games.

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